THE

AMERICAN JOURNAL

OF THE MEDICAL SCIENCES.

SEPTEMBER, 1902.

SOME EXPERIMENTAL AND CLINICAL OBSERVATIONS CONCERNING STATES OF INCREASED INTRACRANIAL TENSION.[1]

THE MÜTTER LECTURE FOR 1901.

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Gentlemen: It is my privilege to bring before the College this evening, as the Mütter lecture, some of the results of a series of experimental observations which relate chiefly to the intracranial circulation as it is influenced by local pathological processes—a subject which, from a bibliographical standpoint, is prominently associated, among others, with the names of Haller and of Astley Cooper, of Fluorens and Majendie, of Key and Retzius, of Leyden, of Althan, of Kussmaul and of François Franck, of Naunyn, Mosso, von Bergmann, of Horsley and Adamkiewicz, of Leonard Hill, and, more recently, of Kocher.

My personal introduction to the subject was due to the interest of Professor Kocher, of Bern, and the investigations as originally undertaken at his suggestion had for their primary objective point the determination whether during an increase in intracranial tension the capillaries and smaller bloodvessels of the brain were dilated from venous stasis, or, on the other hand, whether a condition of capillary anæmia was brought about by such a state.

This question, naturally one of fundamental importance for the proper explanation of the phenomena attending states of cerebral compression,

[1] This paper, in substance, was presented before the College of Physicians of Philadelphia, on December 3, 1901. Its experimental basis will appear in Naunyn and Mikulicz's Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, 1902, No. 9. under the title "Physiologische und anatomische Beobachtungen über den Einfluss von Hirnkompression auf den intracraniellen Kreislauf und über einige hiermit verwandte Erscheinungen."

curiously enough has been the occasion of much disagreement among writers. Its elucidation promised to render many of the problems relative to the symptom-complex of "Hirndruck" much more easy of interpretation.

Not wishing to trespass too far on your indulgence, it is my intention to avoid as far as possible theoretical grounds and the discussion of the technical details of the particular experiments, but to dwell chiefly on their bearing toward operative, clinical, and diagnostic procedures. The brevity of the hour may be an apology for the omission of an historical résumé of the subject.[1]

For the sake of convenience, and in order to avoid confusion from a terminology which has been the occasion of some misunderstanding when speaking of cerebral compression, a division into two groups of the causal processes of such a state will be made. The necessity of such a differentiation, according to von Bergmann, was first emphasized by Griesinger. These groups comprise, on the one hand, processes which occasion, for the most part, a *local compression* of the brain, with a resultant unequal distribution of intracranial tension, in contradistinction to those conditions, on the other hand, which bring about a *general compression* of the central nervous system, with an equal distribution of this tension. On clinical grounds, such pathological processes as a new-growth or abscess, a blood clot, or an abnormal and circumscribed collection of fluid in one part or another of the cerebro-spinal space, illustrate the former condition; that is, subject the brain to the effects of a *local compression*. A foreign body of this sort within the enclosing walls of the skull must be accommodated at the expense of the fluids, vascular and lymphatic, which constitute a not inconsiderable proportion of an otherwise incompressible brain mass. The practical non-compressibility of the brain tissue itself since the time of Alexander Munro's investigations has been unquestioned, and the introduction, therefore, of an intracranial foreign body is only possible through the emptying of the vascular channels in its vicinity. The pressure exerted by such a foreign body, as will be seen, is not transmitted equally throughout the cerebral chamber, and in consequence the circulatory embarrassment in corresponding degree is unevenly felt.

[1] An interesting historical and critical introduction to the subject of the intracranial circulation will be found in Mosso's work (Ueber den Kreislauf des Blutes im menschlichen Gehirn, Leipzig, 1881), and also in Berger's recent monograph (Zur Lehre von der Blutzirkulation in der Schädelhöhle des Menschen, Gustav Fischer, Jena, 1901); and in Leonard Hill's "The Cerebral Circulation," T. & A. Churchill, London, 1896. Ito in his paper (Zur Aetiologie und Therapie der Epilepsie, Deutsche Zeitschrift für Chirurgie, 1899, Bd. lii. S. 489) gives a most complete list of bibliographical references; and more recently von Bergmann (Der Hirndruck und die Operationen wegen Hirndruck, Handbuch der praktischen Chirurgie, 1900, Bd. i. S. 189) and Kocher (Hirnerschütterung, Hirndruck, u. s. w. Nothnagel's Specielle Pathologie und Therapie, 1901, Bd. ix. 3 Theil, 2 Abtheilung, S. 81) have critically reviewed the subject of the circulation in states of compression from the experimental and pathological standpoint.

In the second group of processes, namely, those associated with *general compression*, the intracranial tension may be considered to be equalized throughout the cerebral chamber, and in certain instances, indeed, over the entire central nervous system. Those traumatic cases followed by acute cerebral œdema, which Cannon and Bullard have recently ascribed to alterations in osmotic pressure—cases of acute hydrocephalus, cases of generalized meningitis, of hemorrhage into the subdural spaces from fracture of the base—processes such as these, chiefly ones in which the pressure is transmitted primarily through the fluid contents of the cerebro-spinal spaces, may be given in illustration of states associated with a general increase in intracranial tension.

Now it is not difficult to simulate experimentally the conditions produced by these two forms of compression, and I will endeavor to illustrate as briefly as possible the simple laboratory apparatus which has been employed to this end, as well as the method of direct observation of the cortical circulation during the experiments.

As early as 1811 Ravina, an Italian experimenter, in the attempt to demonstrate the pulsation of the brain within the enclosed cranium, inserted a glass window in the skull for this purpose. Donders subsequently, in 1851, employed a similar method, as did later on Kussmaul and Tenner in their attempt to disprove the so-called Monro-Kellie doctrine that the total amount of blood in the brain cannot vary with an intact skull. Although these observations are often quoted, the method used seems to have fallen into abeyance, and I am unaware that the procedure has been utilized in the study of circulatory changes during compression. It must be confessed that considerable skepticism was felt as to its practicability, but as a matter of fact it was through this method that most important corroboratory evidence of the vascular alterations during the experiments was obtained by immediate observation of the pial circulation.

An ideal window, doubtless, would be one which could be screwed into the trephine opening made for its reception; but as the simple provisional method which was first employed sufficed for all purposes, it was retained. It consists simply of a circular disk of glass with a bevelled edge made to fit into a large trephine opening, and held in place by two screws with flattened ends so bent that they hook under the edges of the opening. Under the trephine opening a small area of dura is removed sufficient in extent to expose the pial vessels overlying a convolution and at the same time an adjoining sulcus with its contained venous radicle. As a rule, also, the trephine opening in the bone has been made so as to overlap the median line, in order to expose the longitudinal sinus, to one side of which the opening in the dura has necessarily been made. (Fig. 1.) The window, though not hermetically sealed, nevertheless answers the same purpose as such a

one, since an increase of tension floats the dura up against the glass in valve-like fashion.

In a great number of my experiments, drawings of the exposed area of the cortex during different stages of compression were carefully made in color, usually with the help of a low-power lens. This was necessary in order to fix in the mind's eye the minutiæ of the smaller vessels and the ground-color of the brain. Only by such observation was it possible to carry a mental picture of the topography of the fine radicles, and consequently to observe accurately the time of their appearance and disappearance from the field of vision. (See Plate, Fig. A.)

For the production of local compression the particular form of apparatus employed was similar to that used by Horsley and Spencer in their experiments, and I believe originally adopted from François Franck.

FIG. 1.

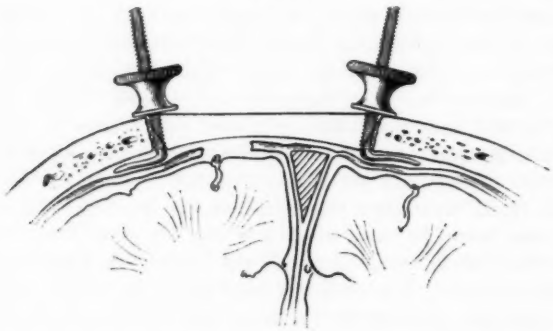
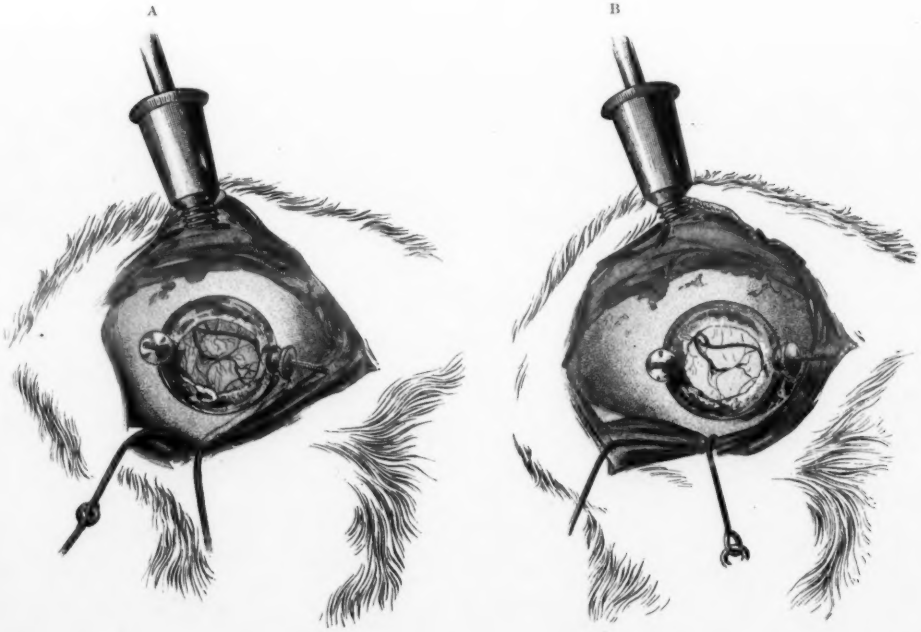


Diagram showing the cranial window in place, its relation to the longitudinal sinus, and the opening in the dura.

It consists of a double canula, the outer metal portion of which can be screwed into a trephine opening in the skull. Into this is inserted a second canula, the outer end of which communicates, by means of a heavy rubber tube of small bore, with a burette of quicksilver. To its inner or cranial end is attached a thin distensible rubber bag. (Figs. 2 and 3.)

By such an arrangement can be told not only the extent of encroachment upon the intracranial space by measurement of the amount of mercury allowed to enter the bag, but also can be told the degree of pressure necessary to evoke certain symptoms and to produce certain circulatory changes by the measurement of the height above the brain of the column of mercury in the burette. Let us suppose that this apparatus has been so introduced that pressure resultant to filling of the bag will act directly against the hemisphere without exciting sensory



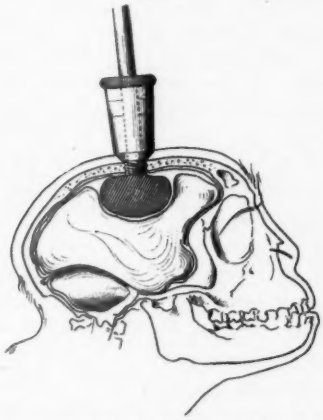
These figures, reduced one-third from original sketches, have lost much in the process of reproduction. Head of Dachshund, showing local compression apparatus and cerebral window in position. *Exp., Feb.* 22, 1901.

FIG. A.—Normal appearance before increase of intracranial tension. The larger vessels should be more of an arterial color.

FIG. B.—Same animal during a period in which local compression against the right hemisphere was carried considerably in excess of the measured arterial (femoral) pressure (local pressure exerted by mercury, 110 mm.; blood-pressure equals 100 mm. of Hg.; displacement of brain produced by bag, at this time, 7 cm.; measurement of brain post-mortem 70 cm.), showing transmission of compression effects to the cortex underlying the window (left hemisphere near parietal eminence). The larger veins remain full of dark non-circulating blood and present the appearance of being broken in places. The arteries are mere threads, and should be barely distinguishable. The cortex is completely blanched.

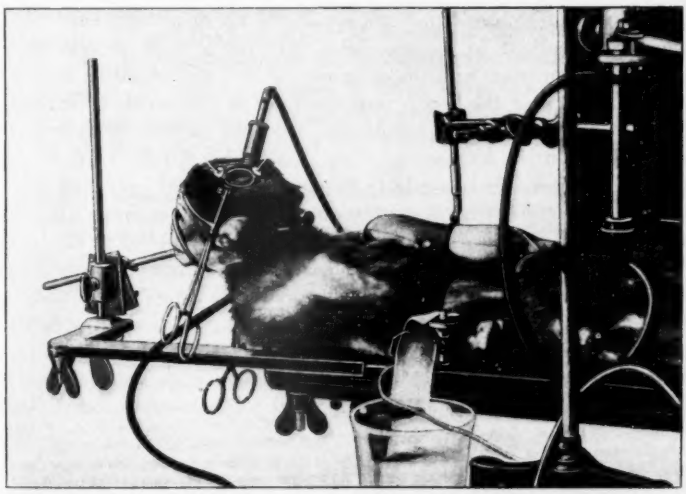
impulses from the stimulation of an interposed dura, a membrane which in the dog is especially sensitive. The first effect which will be

FIG. 2.



Semischematic representation of the method of producing local compression.

FIG. 3.



Photograph showing the cerebral window and local compression apparatus in place during an experiment.

seen to follow upon a moderate degree of compression will be a slight dislocation of the entire brain mass and a distinct widening of the

smaller venous radicles exposed under the window. They at the same time become darker in color, so that their differentiation from the arteries is no longer as difficult as it is in the normal state. As the size of the foreign body increases, this venous stasis becomes apparent at remote portions of the brain, and it can be readily made out in the eye-grounds, where it seems usually to be more marked on the side of the compromising body. If the distention of the bag is further increased the intracranial tension in its vicinity finally reaches that of capillary pressure, at which time the convolutions of the brain not only in the neighborhood of the foreign body, but if the process is still further advanced even in remote parts of the hemisphere, abruptly lose their rosy color and become blanched. (See Plate, Fig. B.) The veins meanwhile remain filled with dark blood, whose means of escape seems to have been cut off, as it were, at both ends. Now, such a condition of anæmia of the hemispheres may be brought about, in the dog at all events, with but little evidence of the so-called major symptoms of compression. There usually is some slowing of the pulse and alteration in respiration, but the tolerance which an animal shows toward such an interruption of circulation in the hemispheres evidences the fact that the medulla has largely escaped from the effects of the local compression.[1]

Thus, as Horsley and Spencer, Hill, and others have emphasized, the brain does not transmit pressure equally in all directions, and so the symptoms resultant to a compression force of a local process vary greatly in accordance with the proximity of the compromising body to the vital centres of the fourth ventricle or to its remoteness from them. This rule applies to local pathological processes as well as to experimental ones in the laboratory. The counterpart, furthermore, of the laboratory experience is regularly seen on the clinical operating-table during the procedure of the elevation of a lobe of the brain such as that necessary for the removal of the Gasserian ganglion. The narrowing of the intracranial space resultant to the extensive compression and elevation of the temporal lobe at such times is productive merely of a slight slowing of pulse, with compensatory rise in blood-pressure, due to a condition of intracranial venous stasis. Such a degree of compression as is necessary to expose the ganglion, if transmitted to the medulla, would naturally be rapidly fatal. If, therefore, in clinical

[1] The degree to which a downward dislocation of the hind brain through the foramen magnum may account for this escape cannot be absolutely determined. That it may play some part in certain animals with conical skulls, such as rabbits and dogs, is quite possible, and attention has been called to this fact by Hill. On frozen sections of brains which have been hardened *in situ* under such circumstances it can be readily seen that the medulla may be dislocated to a considerable extent into the spinal canal. A similar dislocation I have seen to be present in slight degree in a human brain in a case in which a cerebellar cyst had finally caused fatal compression.

states some specialized centres or systems of neurones are not compromised by such a local compressing force, evidence of whose presence would thus be afforded by paralyses of one form or another, the cerebral chamber may harbor a foreign body of one-tenth to one-eighth the size of the brain without necessarily giving an indication of its presence and far from producing any of the so-called characteristic or major symptoms of compression. The venous stasis, however, which we have seen to be caused by such a condition naturally may account for some degree of apathy or stupor which usually accompanies it, and also may serve to explain the early congestion and widening of the veins of the fundus—the primary stage of the "Stauungspapille" of the Germans—in accordance with the original view of von Graefe. The symptoms produced by conditions of local compression, consequently, in so far as they are secondary to circulatory alterations, can be seen to be dependent entirely upon the position of the foreign body, what areas are involved in the local anæmia, and how far-reaching are the congestion effects.[1]

Owing to the irregularity, therefore, of these symptoms which this local method called out, and inasmuch as the data which we chiefly desired concerned the accurate degree of compression to which the *medulla* was subjected during the production of given symptoms, it was determined to employ some method of producing a general and equalized means of compression. In this way alone did there seem to be a possibility of accurately comparing the distinctive and peculiar symptom-complex of "Hirndruck" with the local degree of compressing force about the bulb. Such a "general compression" was produced experimentally by allowing normal salt solution, warmed to body temperature, to enter the cerebro-spinal space through a canula screwed into one part or another of the skull or in the lamina of one of the vertebræ. As a matter of

[1] According to the most recent view of v. Bergmann, if we interpret it correctly, all the symptoms of cerebral compression ("Hirndruck" in his sense) are due to the passive venous congestion which, as we have seen, is brought about by the pressure exerted by a foreign body. He believes, furthermore, that this pressure is transmitted equally over the entire central nervous system through the medium of the cerebro-spinal fluid, and consequently for equal degrees of compressing force, even in the case of a circumscribed body, that symptoms practically of equal severity would be evoked. With this view my experiments are not entirely in accord. It is possible that in some cases of local compression the escape of cerebro-spinal fluid by its natural channels may be interfered with owing to the dislocation of the brain from its normal position and the consequent obstruction of these channels of exit. Under circumstances such as these it is true that the local compression effects may be more evenly and widely distributed. In the majority of cases, however, this does not seem to be the case, and severe compression effects may be felt locally, and little, if at all, in remote territories where measurement shows that the tension of the cerebro-spinal fluid may be but little raised. Although agreeing in general with some of the principles brought forward by Adamkiewicz, a dispute in terminology over "Hirndruck" versus "Druck im Gehirn," seems rather meaningless. The writer inclines to the generally accepted view that the phenomena of compression are responsible to circulatory changes rather than to direct alterations in the nerve-tissue elements themselves from the compromising body as Adamkiewicz claims. In the states of general compression, in the sense in which we have used the term, conditions exist analogous to those claimed by v. Bergmann for all states of compression, but the process, however, must go on beyond one of venous stasis to one of vascular anæmia before the terminal symptoms of compression are produced.

fact, it was found to be immaterial, as far as the production of given symptoms was concerned, over what part of the central nervous system the fluid was primarily introduced, whether over the hemisphere, cerebellum, or cord. This fluid was allowed to enter through a rubber tube connected with a pressure flask, and would spread to all parts of the cerebro-spinal space. The tension under which it stood was regulated by the degree of elevation of the flask, and this was recorded at the same time on a revolving drum by the insertion of a mercury manometer in the course of the tube, in exactly the same way as the arterial pressure was recorded. Thus the blood-pressure and intracranial tension were written from the same abscissa, as is shown in the given chart.[1] (Fig. 4.)

Now there are certain objections which have been raised against the experimental production of an increase of tension by this method. In the first place, according to Adamkiewicz,[2] such a rapid escape of the compression fluid would take place by the normal channels of exit from the cerebro-spinal space that there would be a resultant overfilling of the right heart. Furthermore, it is objected that this escape of fluid would be incompatible with the continuance of an equalized distribution of pressure or one which is truly represented by the height of the pressure-flask.

As a matter of fact, measurement of the amount of fluid lost shows that it is not great, from 60 c.c. to 100 c.c. during a long period of compression—possibly of half an hour—which certainly is not enough to embarrass the cardiac action in the slightest degree. From many observations which I have made, however, there can be no doubt, in agreement with Adamkiewicz, but that there exists very free communication between the cerebro-spinal space and the longitudinal sinus. The original hypothesis offered by Key and Retzius, that the Pacchionian granulations subserve some purpose of filtration, can hardly be entertained; for though the points of exit of the fluid undoubtedly are in the situation of the *lacunæ laterales* of the sinus, into which these granulations for the most part project, their absence in the infant and in the lower animals would of itself be sufficient objection to this theory.[3]

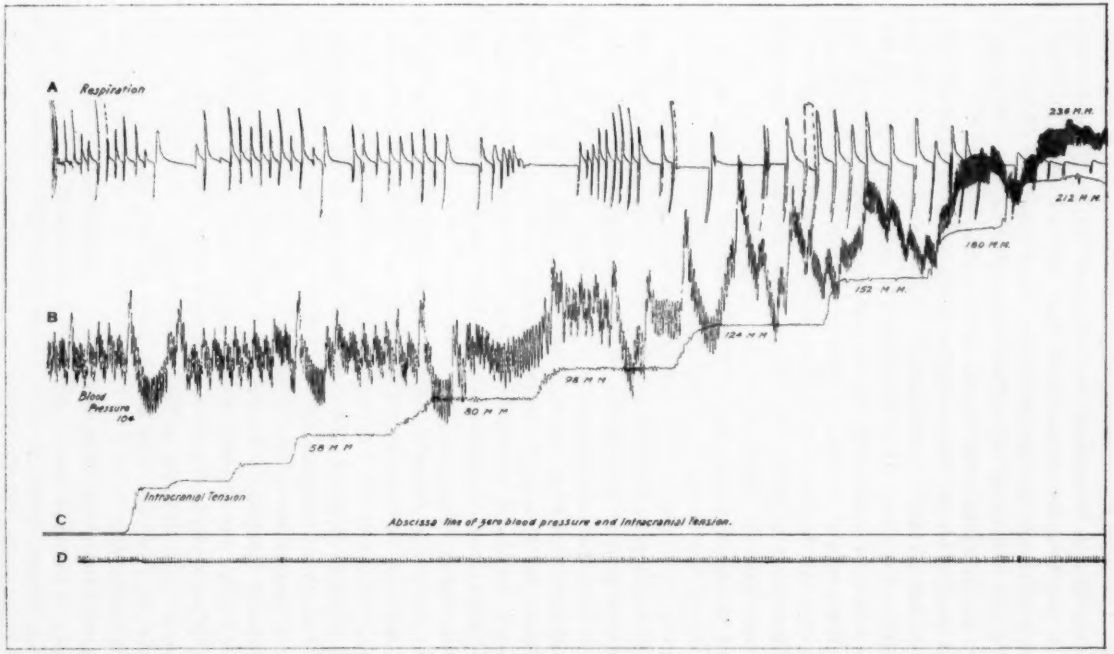
Whatever the nature of these openings may be, or wherever their situation, they certainly are of considerable size and apparently possess

[1] A great number of these charts, whose reproduction is impossible here, were shown for purposes of demonstration. A fragment from a single one of them, taken on a slowly revolving kymograph, accompanies this article.

[2] Neurologisches Centralblatt, 1897, No. 10, vol. xvi. S. 434.

[3] The writer has had the opportunity in Prof. Sherrington's laboratory of performing hemicraniectomy and of turning down the hemisphere for the purpose of stimulating the mesial surface of the brain in the case of several anthropoid apes: gorilla, orang-outang, and chimpanzee. In no instance was there evidence of villæ from the arachnoid projecting into the dura in the neighborhood of the sinus and causing difficulty of separation of the hemisphere at this situation, as is invariably the case in adult man.

FIG. 4.



Fragment of a chart of a "general compression" experiment on a dog. Showing a gradual increase in intracranial tension from zero to 212 mm. of mercury, the blood-pressure meanwhile increasing from its normal level at 104 to 231 mm. of mercury. Note continuance of respiration, absence of vagus effects, etc.

a valvular action. The process is not a mere filtration through a membrane. This is readily shown experimentally by the fact that an injection mass entered, for example, in the lumbar meninges, may be easily made to pass from the cerebro-spinal space into the longitudinal sinus, and thence through the jugulars into the right heart. An injection in the opposite direction by way of the veins never, or but exceptionally and in small amounts, gets into the cerebro-spinal space.

The freedom of the communication of the subdural space with the sinus is demonstrated, furthermore, by the substances which will pass from one to the other. If, as occasionally happened during my experiments with local compression, the rubber pressure-bag containing mercury should rupture, and the globules of quicksilver enter the subdural space, they will be found distributed in the sinus, the jugulars, in the right heart, and throughout the lungs. I have frequently corroborated this experience on the human body by injecting mercury into the lumbar meninges, post-mortem. If the head be inverted, the skull trephined in the median line, and the longitudinal sinus opened, the mercury will be seen to pass readily from the subdural space into the sinus. To illustrate further: on many occasions the attempt was made to produce general compression by the introduction of gases under pressure instead of using the salt solution as above described. Were the gas a non-absorbable one, the animals would quickly die of cardiac air-embolism; and if the jugulars were exposed, bubbles of it could be seen pouring down toward the heart. Exposure of the cervical lymphatics and of the thoracic duct, on the other hand, showed in all instances a complete freedom from gas.

Contrary to the general belief, the longitudinal sinus, in the dog at all events, may be completely collapsed by an increase of intracranial tension. This may play an important part in the prevention of the escape of cerebro-spinal fluid under conditions such as the experimental ones of which we are speaking. It is well recognized in clinical states that the obstruction to the outflow of fluid, be it held back in the ventricles by blocking of the foramen of Majendie, or in the subdural space by inflammatory obstruction of those orifices which lead into the sinus, is especially prone to be associated with pressure phenomena. The escape of fluid under normal circumstances in the adult, whether by the olfactory, optic, or spinal nerve sheaths, plays a relatively inconspicuous rôle.

Waiving these objections, therefore, as without consequence, let us suppose, for example, that the artificial cerebro-spinal fluid be allowed to enter, by means of such an arrangement as has been described, through a trephine opening in the arch of the atlas under which the dura and arachnoid sheaths have been opened. From its proximity to the foramen magnum this would allow the fluid to spread easily up over the

brain by way of the cisterna magna, and the full degree of pressure would be felt in the immediate neighborhood of the medulla. Now, in case of an increase of tension under such circumstances as this, vascular alterations similar to those which we have seen to follow upon the local compression may be observed by direct inspection of the cortex—alterations, however, much more regular in their appearance, and doubtless of corresponding degree, over the entire central nervous system. There occurs, primarily, evidence of stasis, with widening of the venous radicals and a marked color differentiation between the smaller veins and arteries. There also may be detected a distinct bluing of the rosy ground-color of the exposed convolution. Similarly, evidences of venous stasis may, in favorable cases, be made out in the fundus oculi. The eyes themselves become staring and prominent from a retrobulbar œdema, which soon spreads to the lids, and which is due to the escape of the compressing fluid along the optic sheath. This *hydrops vaginæ nervi optici* may accentuate, although I do not believe, in accordance with the theory of Manz, Schmidt, and others, that it is of itself responsible for the symptoms of the "Stauungspapille." It would seem, however, to play some part in the production of an exophthalmos. As the pressure is increased the longitudinal sinus wavers, narrows, and, from being a broad, blue band across the field of the window, thins until it is completely collapsed. Zeigler has brought forth experimental evidence to show that during an increase of intracranial tension the venous pressure in the sinus sagittalis is raised. Evidently, therefore, at some period during an early stage of compression some interference with the outflow of blood is brought about. Complete compressibility, however, of the sinus has been heretofore uniformly denied.

If I may be permitted a slight digression in order to speak for a moment of the anatomy of the venous circulation of the dog's brain, this compression of the sinus and the place where the cerebral veins themselves become obstructed may be made more clear. The large venous radicals which gather the blood from the hemisphere (the superior cerebral ones will be taken as a type of all others) come to lie in the sulci, where they are largely protected from pressure. Only in particular places do they depart from this protection in order to pass over the convex surface of a convolution. This occurs regularly at the superior longitudinal convolution, which they cross before emptying into the sinus sagittalis. At this point the veins seem to be especially subjected to the effects of compression. They enter into the sinus, for the most part, obliquely through long slits in the dura, which possess a valve-like action, so that an increase of tension in the sinus is not transmitted to the veins, and consequently an injection into the sinus, as we have stated, will ordinarily not pass back into them. The superior cerebral veins, furthermore, before entering the sinus, bifurcate, one large branch

entering the diploë to join the diploëtic sinus, the other branch continuing on into the sinus sagittalis. If the sinus becomes compressed the escape of blood may take place in large amount by way of the diploë. These diplöetic sinuses in the dog are very regularly and symmetrically placed,[1] and may accommodate, presumably, the entire bulk of the venous blood from the hemispheres. Although much more developed in canine skulls, presumably sinuses of similar import exist in many cases in the calvarium of man, and under conditions of increased intracranial tension the venous pressure transmitted to them may be greatly increased. It is the experience of all operators to have encountered cases in which a craniotomy has met with an almost uncontrollable hemorrhage from the diploëtic veins. In some cases of tumor, for example, with an associated great increase in intracranial tension, the resultant increase of pressure in the veins may be so great that the bleeding from the diploë is checked with difficulty and only by plugging the openings in the bone.

Let us return once more after this digression to our experiment. During this early period of venous stasis, clinical indications of intracranial mischief doubtless would be present not only from the evidence furnished by the dilatation of vessels in the eye-grounds, but also from the mental drowsiness, apathy, or stupor which this amount of increased tension would produce. The major and characteristic symptoms of compression, however, are not evoked until a later period. As can be seen by comparison with many of these charts before you, the pressure of the fluid against the brain may be increased to the point of its equalling blood-pressure before any symptoms referable to the centres in the medulla may be called forth. Doubtless the pre-exising venous stasis may in some instances be productive of a vagus irritation, with some slowing of the pulse, indicating a nutritional disturbance of the vagal centre; but experimental evidence goes to show that the flow of blood through the vascular channels under these circumstances of stasis is no less than before, the increase of venous pressure being carried back so that the blood flows through the vessels as it might through rigid tubes. It is not until the degree of compression approximates that of blood-pressure—that is, until a condition of anæmia is approached or produced—that the major features of the pressure symptom-complex are called forth. It has usually been stated by investigators that such a degree of compression, if held, will occasion the death of the animal. The actual sequence of events does not seem to have been appreciated, for the reason that there has been no comparative measurement of the blood-pressure and the degree of intracranial tension in so far as the effects of the latter are felt in the region of the medulla.

[1] Drawings of these sinuses are given in the Mittellungen aus dem Grenzgebiete der Medizin und Chirurgie. Loc. cit.

The direct examination of the cortex at this period of equalization of blood-pressure and intracranial tension shows, as would be expected, an abrupt blanching of the exposed convolution. Its rosy color becomes of a grayish-yellow hue, and though the pulsating arteries themselves may be seen against this pale background, and the dark-blue veins in the sulci remain filled with blood, little if any circulation presumably passes between them. A corresponding degree of anæmia under these circumstances of "general compression" is present in the medulla, and the centres are poorly, if at all, supplied with blood.

Now, the usual consequence of this condition is not death, as commonly stated, but a stimulation of the vasomotor centre, which occasions a rise of blood-pressure sufficient to overcome the high intracranial tension. At the same time, through the cerebral window the rosy color can be seen to return again to the blanched convolution. The centres are once more nourished. Respiration, which may have ceased, is resumed, and the circulation by this regulatory mechanism has become re-established. If, after such a readjustment has taken place, the intracranial tension be once more increased and carried up to the point of again producing anæmia by exceeding the arterial tension, the blood-pressure once more rises, in its turn, to the point of exceeding the intracranial tension; so the process may be continued until the arterial pressure be forced to two or more times its normal level. The reaction is illustrated, in one way or another, in all of these charts, in some instances the pressure being held for a time as high as 250 mm. of mercury without evidence of vasomotor failure. As can be seen, on diminution of the tension the exact reverse of the process takes place, the blood-pressure taking a drop of a corresponding number of millimetres of mercury, always tending to remain, however, at a level which prevents the occurrence of anæmia at the bulbar centres.

I have endeavored to describe this reaction in its simplest form, and before speaking of the many variations which occur, let us see what is the physiological action of this response. The mechanism which calls it into play is not a difficult one to demonstrate in our animal experiments. If, in the first place, by division of both vagi we throw out of action their influence on blood-pressure and pulse-rate, and so eliminate the inhibitory action which stimulation of the vagal centre otherwise would occasion, the blood-pressure and the degree of intracranial tension, as the charts show, follow one another with even greater regularity than before. The vagus response, indeed, under other circumstances is apt to mask this simple vasomotor reaction.

If, again, the spinal cord be divided above the level of departure of the sympathetic nerves to the abdominal viscera, and thus the vasomotor control of the great splanchnic area be lost, and then cerebral compression be made, only the vagus effect will be produced, with a

slowing of the pulse, but with no rise of blood-pressure whatever. If both vagi and cord are divided there is no alteration whatever in either pulse or blood-pressure on increasing intracranial tension.[1]

Similarly on cocainizing the medullary centres, and thus in another way throwing out of action both the vagus and vasomotor centres, the blood-pressure will immediately fall, and an increase of intracranial tension will not occasion the usual rise in blood-pressure—at all events, not until the cocaine effect has completely worn away. [A series of charts are here reproduced (Figs. 5–9) to show this reaction.] That

FIGS. 5-9.—Charts from a compression experiment on a rabbit (April, 1901), showing the effects of cocainization of the medulla in counteracting the usual blood-pressure response. Charts to be read from left to right. They are considerably and unequally reduced in size. *A*. Respiration which, after cocainization of medulla (Fig. 2), is continued by artificial means. *B*. Blood-pressure from femoral. *C*. Zero abscissa for both blood-pressure and intracranial tension The line is written by the pen recording the manometer level of the latter. *D*. Time.

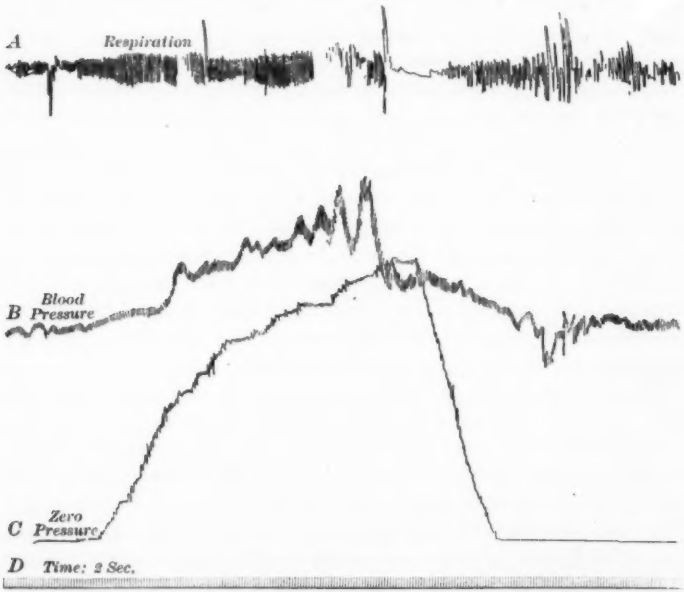


FIG. 5.—General compression experiment on a rabbit. Shows normal compression reaction. On a gradual increase of intracranial tension (*C*) to exceed the original arterial tension (*B*) the latter rises sufficiently to overcome the former. The pulse-rate is not affected, and the respiration continues. (Note that the pen recording intracranial tension (*C*) writes about 1½ cm. ahead of the blood-pressure pen (*B*).) On lowering the pressure flask to zero level the blood-pressure returns to its normal level.

[1] Charts illustrative of these reactions have been given in the Johns Hopkins Hospital Bulletin, 1901, vol. xii. p. 290.

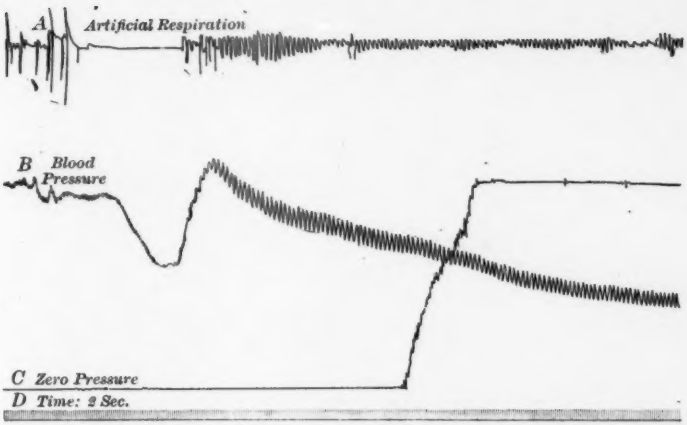


FIG. 6 shows effects upon blood-pressure in the same animal of an injection of cocaine into the neighborhood of the fourth ventricle—an immediate paralysis of respiration, with fall in blood-pressure and rapid pulse. Artificial respiration momentarily checks the blood-pressure fall. It, however, gradually drops to its paralysis level (see Fig. 7, *B*). An increase of intracranial tension (*C*) from its zero level to exceed the level of falling blood pressure is accompanied by no response on the part of the vasomotor centre. (This figure is reduced considerably more than the following ones).

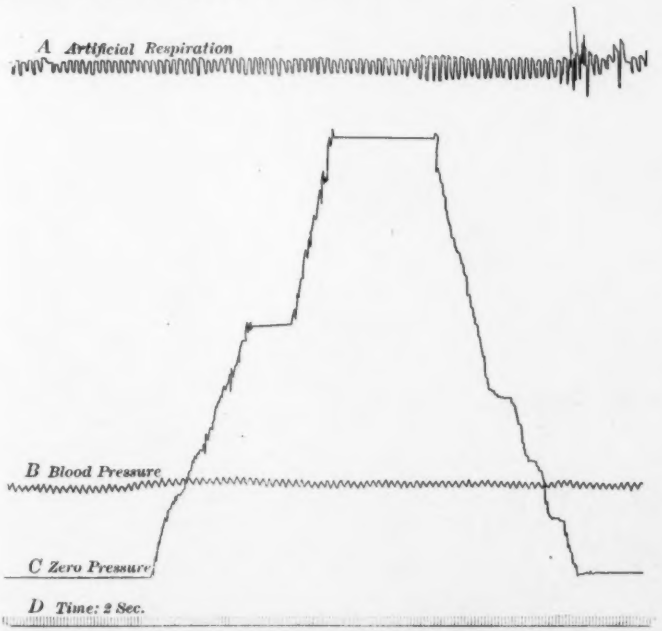


FIG. 7, showing blood-pressure (*B*) at its "paralysis level," 26 mm. of mercury, owing to vasomotor failure due to cocainization of centre. A great increase of intracranial tension (*C*) equal to 172 mm. of mercury does not affect blood-pressure to any appreciable degree.

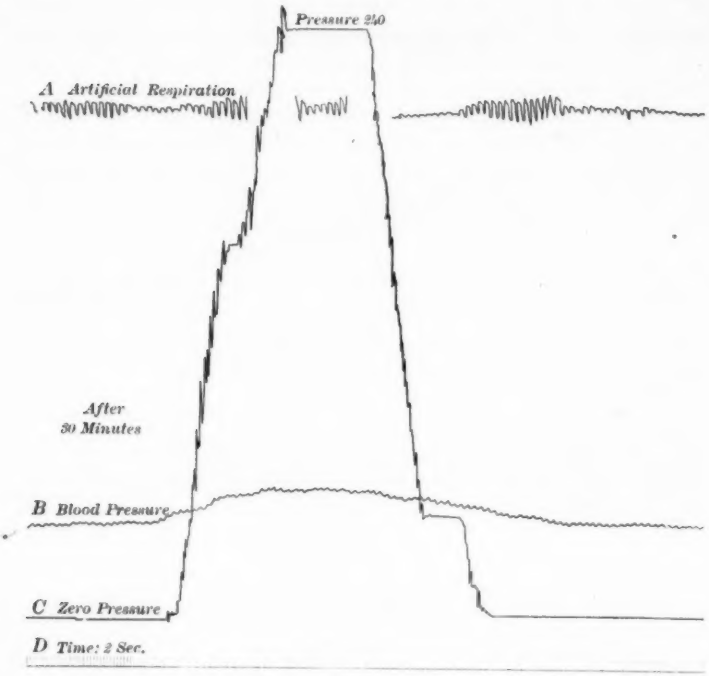


FIG. 8.—Same animal one-half hour later. Slight response on part of blood-pressure (*B*) to great increase of intracranial tension (*C*).

the mechanism is largely due to the vascular control of the great splanchnic field can be readily appreciated during an experiment by the exposure of a loop of intestine. The mesenteric and intestinal vessels can be plainly seen to diminish in calibre during the great rise in blood-pressure, and to dilate again with the fall that occurs in association with the release of the brain from the compression.

Now there are certain irregularities of interest, but of which I have avoided mention lest they confuse the description of this main underlying reaction. If, for example, the intracranial tension is *suddenly* brought to the point of equalling or of exceeding the blood-pressure, there ensue evidences of a pronounced vagus stimulation, usually with a complete checking or "Stillstand" of the heart, lasting at times over a period of thirty seconds and often associated with the production of spasms of the Kussmaul-Tenner type. If the pressure is held, however, there is finally a gradual release from this extreme vagus inhibition, the heart-beat slowly returns perhaps to its normal rate, and the blood-

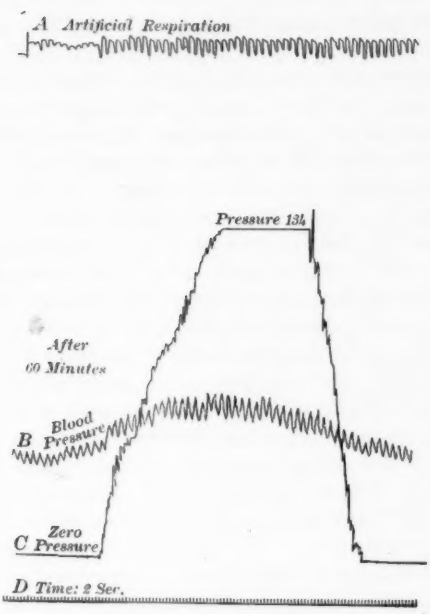


FIG. 9.—One hour later. Cocaine effects wearing away. Blood-pressure response to increase of tension more apparent.

pressure gradually ascends to or above its necessary level. Under conditions which presumably correspond more closely to the majority of those occurring in clinical states, in which there is a very much more gradual increase of tension, such a pronounced vagal irritation may be avoided, and the rise of blood-pressure will occur in anticipation of that degree of compression which would otherwise equal or exceed it, and so bring about a condition of anæmia of the bulb. Thus in many of the observations represented by these charts, by slowly increasing the intracranial tension, even though it ultimately be carried far above that corresponding to the original level of the blood-pressure, we have been able to avoid (as shown by the curves and as corroborated by the view through the cerebral window) any period of circulatory interruption in the medulla. (See Fig. 4.) Only exceptionally, however, has it been possible to obtain this vasomotor response in a fresh animal without sufficient circulatory disturbance in the neighborhood of the bulb to produce some slowing of the pulse and respiration. Usually, when the compression is occasioned with comparative rapidity (from eight to ten minutes, for example) symptoms are produced that are the experi-

mental counterpart of the deep, stertorous respiration, slow vagus pulse, and high blood-pressure which we see clinically in corresponding cases.

Observations which we have been enabled to make on an occasional case of fracture of the base in which hemorrhage has occurred into the subdural spaces have given us exact clinical corroboration of the experimental reactions. If in these cases it has been impossible to relieve the intracranial tension by the removal of a wide area of bone, a *circulus vitiosus* of symptoms is established in the following way. There is, in the first place, an increase of intracranial tension from the effused blood. When this becomes sufficient in amount to embarrass the circulatory conditions at the medulla the familiar clinical picture is displayed, with the slowing of pulse and respiration, and the high, bounding, incompressible, peripheral artery, whose tension can be measured, at times, almost at 300 mm. of mercury. This rise in blood-pressure, which under all circumstances is a conservative one, for the furtherance of a proper nutritional supply to the vital centres, here suffices merely to increase the intracranial bleeding, and thus in a still greater degree to increase the tension which has primarily evoked the response. Under such circumstances the vasomotor centre rapidly fails.

Another characteristic, which many of these blood-pressure curves show when the vasomotor centre is straining to overcome the condition tending to produce bulbar anæmia, is that of a rhythmic or wave-like variation in pressure—the well-known Traube-Hering waves—the causation of which has been the occasion of much dispute. In these instances it can be seen that the crests of the waves rise above and their hollows fall below the level of the intracranial tension. Examination of the circulatory condition of the brain through the window at the time of their production shows that an interval of cerebral anæmia corresponds to the low-pressure stage of the wave, whereas a condition of active capillary circulation rides on their crests. These circumstances are well shown on many of these charts taken during stages of general compression experiments. When the compression exceeds the blood-pressure anæmia ensues; the vasomotor centre is stimulated; the blood-pressure, rises in these cases higher than is necessary to readjust the circulation; the bulb is well supplied with blood—perhaps too well—and is hyperæmic; the stimulated centre relaxes to too great a degree; the arterial pressure falls below the level at which blood can enter the compressed vessels; the medulla once more becomes anæmic. Thus the centre is again stimulated, and so the process is repeated indefinitely. These rhythmic waves, furthermore, in many of these charts can be seen to follow the general level of blood-pressure as this is raised or lowered by alteration in the degree of intracranial tension.

It is quite possible that these rhythmic activities are nothing more than an exaggeration of the normal rhythmic waves of blood-pressure which are known to take place under a variety of conditions. Furthermore, as has been stated, the primary object of this elevation of blood-pressure seems to be for the continuance of nourishment to that most important of all centres, the respiratory one. A period of anæmia such as occurs during the low-pressure stages of the Traube-Hering waves leads promptly, as a rule, to the cessation of respiration. As long, however, as the vasomotor centre keeps the blood-pressure higher than that of intracranial tension, and at a point sufficient to keep the medulla supplied with arterial blood, respiration continues. Thus accompanying the crests of the waves of blood-pressure can be seen respiratory efforts, which assume more or less the character of the Cheyne-Stokes type of respiration. The period of shallow respiration or of actual apnœa accompanies that stage of the wave which is associated with cerebral anæmia. Such a periodicity of respiration, as we shall see, can be demonstrated clinically in association with waves of blood-pressure; shallow or passive respiratory efforts accompanying the low stage, deep and snoring respirations the rhythmic periods of its elevation.

The length of time experimentally during which these reactions can endure is considerable, even under the extreme degree of elevation—over 200 mm. of mercury from a level of 90 or 100 mm., in many cases—to which we have pushed the blood-pressure. These extraordinary responses under clinical conditions doubtless are called out only in rapidly fatal cases, such as those of basal hemorrhage with fracture, as has already been instanced. The more slowly the intracranial space is compromised probably the longer will the response hold. In the experimental as well as in the clinical cases, unless relief is given, a period comes at which the vasomotor centre weakens, loses its grasp on the great splanchnic field, the blood-pressure falls below that of the intracranial tension, the respiratory centre is no longer nourished, and the individual dies with a low blood-pressure and rapid heart, whose action may persist for hours, provided that the respiration is continued by artificial means.

A striking instance of this kind came under my observation a short time ago—one which illustrates just such a terminal stage and also the danger of any further increase of tension during the critical period of cerebral compression with bulbar involvement. The patient entered the hospital in a condition of profound stupor, with a subnormal temperature, a snoring respiration, a heart beat of fifty to the minute, a high-tension pulse (unfortunately it was before the days of a practical blood-pressure apparatus, and I cannot give the exact figures), and a "Stauungspapille"—all indicating such an increase of intracranial tension that the bulb was in the condition bordering on the so-called

"Lähmungsstadium" of the Germans. With evidence of an old otitis media as a localizing indication, an operation was immediately performed. The abscess was difficult to find, inasmuch as it had no apparent communication with the diseased temporal bone, and in the course of our search, during the elevation of the temporal lobe which was suspected to harbor it, the respiration ceased. The medullary centre, evidently suffering already from compression effects, could not endure this additional burden. On immediately releasing the pressure, and after some artificial respiratory efforts, spontaneous breathing returned. On resuming the search the abscess was finally located and evacuated, but the respiration during the procedure had again ceased. This time, in spite of artificial respiration, spontaneous breathing was never resumed, though every possible means of reawakening it by sensory stimulation and otherwise was resorted to. The characteristic rapid pulse, of low tension, indicating a vasomotor failure, had appeared, and at this low level of blood-pressure the heart continued to beat for *twenty-three hours*, during which time artificial respiration was continued. Though a careful gross and microscopical examination was made of the medulla of this case by Dr. Paton, no lesions worthy of note could be detected. Similar instances of respiratory failure anticipating the cardiac failure by many hours have been reported by Victor Horsley, Macewen, and Hudson.

It has been heretofore pointed out that in death from a fatal increase of intracranial tension the arrest of respiration precedes that of the heart. Back of the respiratory failure, however, in the majority of cases, and responsible for it, if we may judge from experimental evidence, stands the collapse of the vasomotor regulatory mechanism, which up to this time has held the blood-pressure at a height sufficient to overcome the abnormal extravascular tension at the medulla. Prompt surgical relief, with a wide opening of the calvarium, may save life even in desperate cases with pronounced medullary involvement. Thus an extensive craniectomy may completely, though temporarily, ward off the serious attacks which accompany the increasing tension of an intracranial growth. In acute cases, on the other hand, as in those of hemorrhage accompanying basal fracture, I believe that in the majority of instances the pressure symptoms may be completely and permanently warded off.

Two years ago there was brought to the hospital a small boy who had fallen, as a child falls, head downward, from a third-story window to a basement below. His skull had been fissured in all directions, like a fallen egg-shell, and when first seen he was nearly in the "Lähmungsstadium" of compression—comatose, with a slow pulse of very high tension, a Cheyne-Stokes respiration, and convulsive spasms of the Kussmaul-Tenner type, resembling those seen during the period of cere-

bral anæmia in the experimental conditions. An immediate operation—laying back the tense scalp by anterior and posterior flaps, such as are made on the autopsy-table, with elevation of large plates of depressed bone and the removal of many fragments—resulted in the immediate relief of all bulbar symptoms. The brain itself, however, was so hemorrhagic and disorganized that there seemed to be little hope of its ever returning to a functional condition. For three weeks the patient lived the life of a spinal animal, unconscious and with absolutely no spontaneous movement, but with normal pulse and respiration and normal visceral reflexes, so that he would swallow when liquid was put in the back of his mouth. Not until twenty-one days of this absolutely passive state had elapsed was there evidence of the return of some cerebral activity by a slight motion in one hand and the occasional feeble elevation of the eyelids. He ultimately made a complete recovery, but had narrowly escaped at the time of operation the period of vasomotor paralysis.

It must be remembered, however, that the sudden removal of pressure from the brain when the blood-pressure has been forced to considerable heights may be followed by a paralysis instead of a release from the major compression symptoms. The occasion of this is readily brought out by post-mortem examinations, which, under such circumstances, oftentimes discloses a brain and medulla of a uniform cherry-red color, from the widespread extravasation of blood due to the multiple rupture of the minute bloodvessels. The external supporting pressure of the high intracranial tension has been suddenly removed, leaving the internal or intravascular pressure too great for the strength of the vessel walls.

An opportunity was recently offered, in Dr. Osler's wards, to follow through the paralytic stage of compression a patient for the relief of whose condition operative interference had been refused. A brief relation of the clinical observations made in this case may more closely bring into line the bedside observations with some of these experimental experiences.

The patient was brought to the hospital with the history of indefinite mental symptoms which had extended over a period of some weeks. He had become somnolent and apathetic, and had gradually lost the use of the entire left side. Examination showed an individual in stupor who would not reply to interrogation, but could be roused sufficiently so that he would show his tongue and open his eyes. The eyeballs themselves seemed somewhat prominent and the lids somewhat congested. A bilateral choked disk of advanced degree was present. There was apparently complete motor paralysis of the left arm and leg, with some anæsthesia. The right leg was also paralyzed, and the increased muscular tonicity of the right arm showed that paralysis might

be expected there. There was no acceleration of the pulse (80). The respiratory rhythm was unaltered. There was no abnormal increase in blood-pressure. It was evident that some local process was present in the right brain, the tension from which was transmitted in sufficient degree to the opposite hemisphere to cause profound alterations in circulation, as shown by the pressure paralysis and by the change in the fundus oculi. No symptoms, however, had yet given evidence of any circulatory disturbance in the remote situation of the bulb. At the end of five days, with a gradually deepening stupor, the medulla began to show signs of involvement, chiefly as an increased tension of the pulse. As in the experimental conditions, when the intracranial tension about the bulb is slowly and gradually brought to the neighborhood of the blood-pressure the latter may rise to counteract its effects, without the production of the slow vagus pulse and deliberate respiration almost invariably seen in more rapid processes.[1]

At the time of this beginning period of bulbar involvement a needle was inserted in the lumbar meninges and the pressure there registered —as I believe the pressure should always be recorded in case of lumbar puncture—by connecting a long glass rod of small bore with the needle, and by measuring in this way the height to which the column of fluid rises. It should normally equal the venous blood-pressure, about 4 mm. of Hg. In this particular case the fluid rose to a height corresponding to 44 mm. of mercury. Evidently the local increase in tension was transmitting its effects, although in diminished degree, to the remotest part of the cerebro-spinal space. The blood-pressure meanwhile measured between 180 and 200 mm. of mercury. By means of the blood-pressure apparatus, furthermore, the rhythmic waves of arte-

[1] For the proper estimation of gradual alterations in arterial pressure in clinical cases reliance should not be placed alone on the palpating finger any more than that the hand should be depended upon for the determination of a patient's temperature. The arterial pressure should be spoken of in figures, just as is the temperature and pulse-rate, and not in the loose way generally employed, in which we describe it as "weak," or "thready," or "bounding," etc., as the case may be. For this purpose we should have a plotted record of the pressure, since it is of far greater importance in all conditions, surgical and medical, than is the pulse-rate which we so studiously record. There are to-day simple forms of apparatus which may be easily set up and employed for practical purposes. The particular form which has been introduced into the wards of the Johns Hopkins Hospital was demonstrated. It requires no particular familiarization for its satisfactory employment. It represents the Riva Rocci apparatus in its simplest form, and consists of a distensible rubber armlet made to encircle the arm or leg, and which may be inflated until the peripheral pulse is shut off. The degree of tension necessary to accomplish this is registered on a simple mercury manometer inserted between the armlet and the pump used for the inflation. It is true that this apparatus gives us simply a measurement of systolic pressure, and as Brush and Howell have recently emphasized, such a systolic pressure does not represent the mean blood-pressure. As a matter of fact, however, only in exceptional instances is there an inequality in the variations of systolic and diastolic pressures so marked as to be of clinical import, and the simplicity of this apparatus as a means of obtaining one of them, the systolic, renders it of inestimable value as a clinical "instrument of precision." The Mosso apparatus registering diastolic pressure, the Gärtner tonometer, and the Hill apparatus with spring manometer, all possess for clinical purposes insurmountable disadvantages.

rial pressure could be easily made out. If the encircling band of the apparatus about the upper arm was held at a sufficient degree of tension to collapse the brachial artery, and so to obliterate the radial pulse at the wrist, after a few moments the pulse would appear for a short time and then once more fade away, but only to again reappear in this wave-like fashion. A similar and associated periodicity of respiration, furthermore, was seen to be taking place. Coincident with the crests of the blood-pressure waves occurred active periods of respiration, with at the same time a wide dilatation of the pupils and the swinging out of the eyeballs in conjugate deviation toward the side of the lesion. Coincident with the periods of low blood-pressure occurred the shallow respiratory action, and at such times there was usually an asymmetrical condition both as regards the size of the pupils and the position of the axes of the globes.

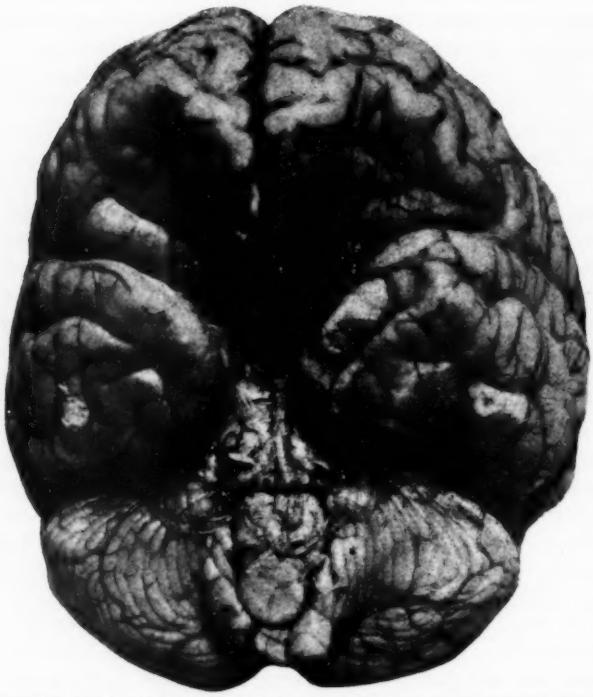
Other and detailed analyses of symptoms I will pass over, merely wishing to draw an analogy with the experimentally induced processes of like nature. After about twelve hours of the high-tension pulse the vasomotor centre began to show signs of failure, the blood-pressure gradually dropped, the tension of the cerebro-spinal fluid in the cord fell considerably, as was told by a second lumbar puncture,[1] and the patient finally died from a respiratory failure, leaving the rapid-running, uncontrollable pulse of complete vasomotor relaxation to end the story.

Clinical corroboration of these experimental data which have been given might be drawn from a multitude of cases. An instance of the effect of local compression in the near neighborhood of the medulla was given by a recent case of cyst of the cerebellum which was seen in Dr. Osler's clinic. As has been stated, the local compression effects are most pronounced in the immediate vicinity of the compromising body. A growth in the cerebellum consequently is one which is apt to suddenly throw out of action by circulatory embarrassment the regulatory centres in the medulla. This may occur before other absolute evidence of its presence has been given, and in this particular case the diagnosis of an intracranial growth had not been definitely made. From the remote-

[1] Just how much stress may be laid upon the measured tension of the fluid in the spinal meninges for purposes of diagnosis and for the estimation of degrees of concomitant intracranial tension, I am unable at present to state. Certainly in localized intracranial processes it does not represent, as v. Bergmann's theory would indicate, the degree of "Hirndruck," as this case clearly evidences for a general compressing force of 44 mm. of mercury is by no means a fatal one. Presumably the dislocation downward of the brain, and consequent blocking of the foramen magnum, plays a certain part in shutting off the spinal fluid from the effects of tension existent within the cranial chambers. Nevertheless, in the cases of cerebral tumor in which I have performed lumbar puncture, there has been some abnormal increase in the tension of the spinal fluid. In the clinical states representing conditions of general compression, such as meningitis, acute cerebral œdema, subdural hemorrhage, etc., the tension of the fluid in the lumbar meninges more nearly corresponds with the intracranial tension which is producing the compression symptoms.

ness of the process to the ophthalmic veins the vascular disturbances of compression due to venous stasis were not transmitted to the eye-grounds, which were normal in appearance. The patient complained of some occipital headaches, of weakness, and there was some exaggeration in the deep reflexes. A lumbar puncture had been performed, evidencing but slight, if any, increase in the tension of the fluid in the spinal men-

FIG 10.



Photograph of the base of the brain of a case which died from compression effects of a cerebellar cyst. The dotted line corresponds to the imprint of the edge of the foramen magnum A mould of the upper part of the spinal canal is made by the included portion of cerebellum and medulla.

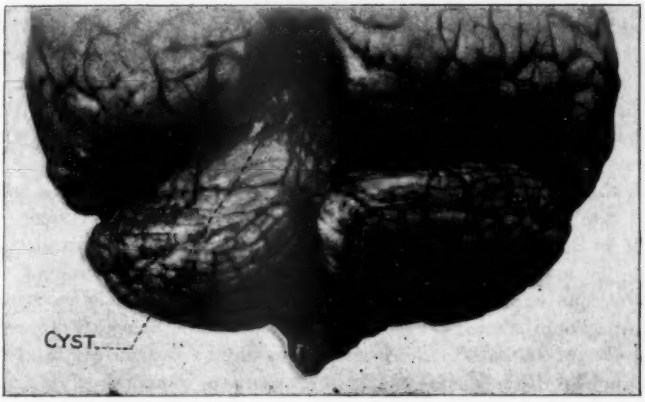
inges. The patient unexpectedly one night passed into the stage of medullary involvement, with loss of consciousness, a slow vagus pulse, stertorous respiration, and a very high blood-pressure, which, unfortunately, owing to the urgency of the case, was not registered, but which was said to have occasioned an almost incompressible pulse. In a short time the vasomotor mechanism gave way, respiration ceased in conjunction with the fall in blood-pressure, and, as in the majority of

these conditions, the heart continued to beat for some time after death had actually ensued.

The accompanying photographs (Figs. 10 and 11) show in this case the marked dislocation downward into the foramen magnum of the medulla, together with a surrounding ring of cerebellum. Whether this dislocation, as has been suggested in the case of lower animals, could allow the medulla in any way to escape from the compression effects, or whether it would actually increase their action, must remain a question of uncertainty.

There are other points of interest concerning which my curtailed time forbids making more than passing mention. The pulsation of the brain, for instance, which, on Mosso's principle of the plethysmograph,

FIG. 11.



Posterior view of same, showing cyst and portion of cerebellar mould of spinal canal.

increases *pari passu* with the increase of intracranial tension, becoming most marked when the pressure from without and the blood-pressure correspond, may give the surgeon at times valuable data. When transmitted to the artificial cerebro-spinal fluid, as in the experiments, it may even exceed in the length of the manometric excursions those of the arterial pulse itself. It is perhaps worthy of note that the character and height of this pulse-wave do not correspond exactly to that of the arterial pulse.

Of interest also is the question of vasomotor nerve supply to the cerebral vessels themselves. Undoubted experimental evidence of their presence has baffled physiological investigation, although the anatomist seems to have already prepared the ground by a histological demon-

stration of their presence. On the clinical side also the existence of such nerves would be favored by instances such as the recurring appearance of paresis or aphasia in cases of Raynaud's disease in association with evidences of local vasomotor spasm of other territories of the body. If, however, one may draw conclusions from the mechanism of the vasomotor centre above described, a mechanism which constricts the vascular fields of the body, thus raising the blood-pressure in order to supply an anæmic medulla with blood, a corresponding narrowing of the cerebral vascular field from the same stimulus would seemingly counteract the conservative object of this mechanism. Contrary to the positive statements of many, substances like epinephrin will blanch the pial vessels over the area of its application, as will occasionally a jet of cold water against the brain or the faradic current used for cortical stimulation. Whatever part, however, the cerebral vasomotor system may play, if such a system there be, it certainly is controlled from centres and by influences other than those which call into play the vasoconstriction in the peripheral and intra-abdominal fields.

From this rather superficial review of the alterations in the cerebral circulation as they are influenced by varying degrees of compression some effects stand out with especial prominence.

In the first place, the venous stasis, which becomes apparent on but a moderate increase of the tension, fortunately gives early evidence of itself in the eye-grounds except in those cases of local compression, in posterior basic meninges, for example, so remotely situated that the compression effects are not readily transmitted as far as the cavernous sinus and ophthalmic veins. Furthermore, local pathological processes, such as are confined to the hemispheres, may be responsible for local circulatory disturbances sufficient to cause a cessation of function of a large part of the forebrain without leading in any way to a corresponding implication of the medulla. When, however, the local process is in the near proximity of, or, if remote, when its effects are so far reaching that the vital centres of the bulb are compromised, the one symptom which with regularity is called forth, and which betokens a serious alteration in the local circulation, is a persisting rise in blood-pressure, which may or may not be associated with a pronounced vagus pulse, with rhythmic alterations in blood-pressure and with a retardation or periodicities of the respiration approaching a Cheyne-Stokes type.

The first and minor symptoms of compression are found in association with varying degrees of intracranial venous stasis, the major symptoms of "Hirndruck," with an approaching capillary anæmia of the medulla.

A REPORT OF NINETY-TWO CASES OF THERMIC FEVER TREATED AT THE PENNSYLVANIA HOSPITAL IN THE SUMMER OF 1901.[1]

By Morris J. Lewis, M.D.,

and

Frederick A. Packard, M.D.,
of Philadelphia.

The 92 cases of thermic fever upon which this report is based occurred in the service of the writers at the Pennsylvania Hospital, Philadelphia, during the extremely hot weather of July, 1901, and includes all those showing a temperature of 100° F. and over, upon admission, and does not include a large number of cases of heat exhaustion. These records, unfortunately, are far from complete, as the extra work thus suddenly thrown upon the hospital staff made it impossible to keep full and accurate notes, the question of treatment being the one of paramount importance. Enough data have been secured to warrant, in our opinion, this report. The amount of work may be appreciated when it is known that 1,000 cases of all kinds were admitted into the wards during the first week of July.

Seventy-six cases were admitted on the first three days of July, 1901, with 19, 26, and 31 cases, respectively. These three days followed three very warm days in June, with a maximum temperature varying between 96° F. and 99° F. The maximum and minimum temperatures of these three days in July were as follows:

	Maximum.	*Minimum.*	
July 1 . .	. 100	82	
" 2 . .	. 102	94	(These days also showed a high humidity.)
" 3 . .	. 105⅕	80	

These records were kept at the Pennsylvania Hospital, and although higher than the official records kept by the Weather Bureau, probably represent more closely the temperature experienced by the great mass of people.

The first case was admitted on June 23d, while from the 6th to the 16th of July no case was admitted owing to a decided fall in the temperature.

The time of day at which the greatest number were received was from 11 A.M. until 5 P.M., the maximum being from 3 to 5 P.M., towards the close of the working day, showing the effect of fatigue and long exposure.

But little time probably elapsed between the onset of the attack and the admission of the patient into the hospital.

[1] Read at the meeting of the Association of American Physicians, May, 1902.

Sex. Thirty-one of the cases treated were females, as against 60 males. This is an unusually large proportion of women, but it should be mentioned that the most severe cases were usually found in men.

Age. The extremes of age of the cases were from seven months to over seventy years, the average being about thirty-seven years. Among the elderly patients might be mentioned one aged seventy years (died, temperature 110⅘° F.), one aged seventy-three years (cured, temperature 108° F.), one aged seventy-five years (cured, temperature 107⅘° F.), and one aged seventy nine years (cured, temperature 108° F.).

Color. Six of the number were negroes, emphasizing the previously well-known immunity of this race.

Nationality. The largest proportion of patients were natives of the United States.

Of the patients showing a temperature of

100° F.	to	and	not	including	101 F°.,	there	were	. . .	24	cases.
101	"	"	"	102	"	"	. . .	8	"	
102	"	"	"	103	"	"	. . .	10	"	
103	"	"	"	104	"	"	. . .	4	"	
104	"	"	"	105	"	"	. . .	4	"	
105	"	"	"	106	"	"	. . .	4	"	
106	"	"	"	107	"	"	. . .	8	"	
107	"	"	"	108	"	"	. . .	6	"	
108	"	"	"	109	"	"	. . .	8	"	
109	"	"	"	110	"	"	. . .	2	"	
110	"	"	"	111	"	"	. . .	8	"	
111	"	"	"	112	"	"	. . .	3	"	
112	"	"		113	"	"	. . .	1	"	
				"High"	. .	. .	. .	1	case.	
				"Unknown"	. .	. .	. .	1	"	
								92	cases.	

No patient with a temperature under 106° F. died, but above this temperature the proportion of "cures" to deaths was as follows:

106° F.	to	107° F.	6 cures	and	2 deaths.[1]	A mortality of 25 per cent.
107	"	108	5 "	"	1 death.	" 15.6 "
108	"	109	7 "	"	1 "	" 12.5 "
109	"	110	1 "	"	1 "	" 50 "
110	"	111	5 "	"	3 deaths.	" 37.5 "
111	"	112	0 "	"	3 "	" 100 "
112	"	113	0 "	"	1 death.	" 100 "

No case with a temperature of 111° F., or over, recovered.

The total number of deaths occurring numbered 13, a mortality of 14.4 per cent. The case recorded as of "unknown" temperature died, and is not included in the above percentage of deaths.

Convulsions. These were noted in 14 cases and were generally of severe type. They were largely tonic in character, and did not resemble those seen in epilepsy or in uræmia, and usually occurred in

[1] These cases, which so inordinately raise the percentage mortality for this class, are detailed somewhat below.

those showing a temperature of 106° F. or over. Retraction of the head was seen in several cases, with or without convulsions, and some showed marked excitement after the temperature was reduced, and struggled violently.

Five of the cases were not convulsed on admission, but showed this symptom after the temperature was reduced by rubbing with ice, with or without bleeding and transfusion. These cases will be spoken of again under the heading "Treatment." Some of the convulsions were unusually severe and tetanic in character. In one case where the temperature only reached 101⅘° F. there were marked tonic spasms of the hands and feet after consciousness was regained.

Mental State. Patients showing a temperature of 106° F., and over, were generally unconscious when brought to the hospital, although one case was unconscious with a temperature of only 101⅘° F., while the term "semiconscious" was recorded for many having the lower grades of temperature. Marked cerebral excitement, even requiring restraint, was seen in a few cases after the reduction of temperature was accomplished.

Pupils. Careful records of the state of the pupils were not kept. In 5 cases the pupils were noted as "contracted" in patients whose temperatures varied from 104⅕° F. to 111⅖° F. In 2 cases with a temperature of 110° F. the pupils were noted as "dilated." Nystagmus, both horizontal and vertical, was noted in 1 case with a temperature of 108° F.

Knee-jerks. The condition of the knee-jerks was examined in 6 cases where the temperatures varied from 101⅘° F. to 110⅘° F. These patients were all unconscious when admitted, and had either retraction of the head or convulsions, which were either tonic and local, or chronic and general. The patient whose temperature was 101⅘° F. was unconscious on admission, but upon a return to consciousness after treatment had tonic contractions of the hands and feet, and showed a marked increase in the knee-jerks, as might be expected from the reinforcement. The remainder of the cases had a total absence of knee-jerks. It is not noted at what time of the patient's stay in the hospital this symptom was noticed, except in 1 case. In this case the temperature reached 110° F., the man was profoundly unconscious and had dilated pupils, there was also retraction of the head, but at no time were there any true convulsions. He rapidly responded to the ice-rub and did so well that no other treatment was instituted. His knee-jerks were entirely absent on the day of his discharge, three days after his admission. An examination recently made of this patient showed the knee-jerks to be present, although a little less pronounced than usual. A second patient whose temperature also reached 110° F., and in whom this absence was noted, has upon a recent examination showed a return of this reflex.

This case had violent convulsions after his temperature had been reduced by treatment.

A third case, that of a powerful negro, was admitted totally unconscious, with a temperature of 108° F. This man also had general tetanic convulsions coming on after the reduction of temperature following the use of ice, bleeding, and transfusion. On the first day of his stay in the hospital it was found that he had absent knee-jerks. On recent examination of this patient, who still claims to be as powerful as ever, it was found that only the slightest possible knee-jerk could be obtained by reinforcement. His station was good, and he had no other evidence of disease of the spinal cord that could be detected.

It is worthy of note that two of these patients state that since the thermic fever they have been impotent, although before their attack they had been possessed of full vigor.

Urine. The urine was examined in 10 cases, but usually not until convalescence had set in, or the case had been retained in the hospital for several days. In 4 of these cases, with temperatures of 100⅘° F., 107° F., 110° F., and 110⅘° F., albumin in varying amount, with granular casts, was found. In 1 case granular casts, without albumin and in another albumin in small amount without casts was found. The urine record of the case showing a temperature of 110° F., an occasional hard drinker, aged forty-eight years, was as follows: specific gravity 1019, acid, albumin, no sugar, dark granular casts containing blood.

This case was lost sight of until very recently, when an examination of the urine gave the following results: specific gravity 1015, distinctly alkaline, light yellow, cloudy, albumin and sugar negative, triple phosphates, calcium phosphates and amorphous phosphates, making it probable that the thermic fever and not the previous physical condition had been the cause of the renal disturbance. In 2 cases sugar was found, 1 having a temperature of 102⅕° F., which was probably a true case of diabetes mellitus; in the second, with a temperature of 108° F., the "sugar" disappeared a few days after its discovery.

Habits. The previous habits of the patient seemed to have an important bearing upon the result, and to affect the latter unfavorably. In the mild cases no alcoholic history was obtained, possibly from lack of interest in the case or from press of more important work, and all recovered. In the severe cases 50 per cent. of those recovering gave an alcoholic history, and of those who died the same percentage was found. This record as to the use of alcohol was not made in all of the severe cases.

Blood. It was thought that many if not all of the symptoms of thermic fever might be due to dehydration of the blood from prolonged and excessive sweating. According to Vincent (Bordeaux *Thesis*, 1887), Chossat in 1868 first broached the theory that thermic fever was due

to excessive sweating and loss of water by evaporation from the lungs. A considerable number of experiments have been performed upon animals to show the effect of exposure to heat upon the composition of the blood. A few of these may be worthy of citing.

Vincent, in experimenting on animals, found an increase in the hæmoglobin percentage and an augmentation of the number of red cells by 300,000 per cubic millimetre.

Grawitz (*Zeitsch. f. klin. Med.*, 1892, Bd. xxi. p. 465), also by animal experimentation, found the specific gravity of the blood usually progressively increased by sweating, although in a small number of cases there was a lowering of the specific gravity. In 1 case the specific gravity rose from 1040 to 1051.

Löwy (*Berl. klin. Woch.*, 1896, No. 41) exposed rabbits to heat for twenty-four hours and found a diminution of the thickness of the blood and dry residue, but all of the tissues "poor in water." The blood serum, on the other hand, was found to contain less water than normal, and while the specific gravity of the blood was found to be diminished, that of the serum was decidedly increased. He attributes these alterations to changes in the vascular tonicity.

Friedländer (*Fortsch. der Med.*, July, 1897, p. 521), also by experimentation, found an increase in the number of the erythrocytes, still more marked increase in the number of the leucocytes, and an increase of the specific gravity and of the thickness of the serum.

Scagliosi (*Virchow's Arch.*, 1901, Bd. clxv., pp. 15 to 41) placed guinea-pigs in the sun in August and September and found an increase in both the erythrocytes and the leucocytes, especially the latter. He concludes from his experiments that "the blood shows an excessive increase in thickness of all formed elements, and of the neutrophilic cells. In some cases, also, a leucocytosis was present."

We can find but little evidence as to the condition of the blood in human beings exposed to excessive temperatures. Alexander Lambert (Loomis-Thompson, *Amer. Syst. of Practical Med.*, 1898, vol. iii. p. 873) states that in 1896 the hæmoglobin value in 12 cases examined ranged from 85 per cent. to 125 per cent. Leucocytosis was present and the leucocytes contained pigment. The changes described were present in those whose temperatures did not reach 105° F., as well as in those with higher temperatures.

Owing to the small amount of exact knowledge as to the condition of the blood in human victims, and owing to our thought that in changes in the blood an explanation of at least some of the symptoms might be found, it was our intention to pay particular attention to the condition of the blood in any cases of thermic fever brought to the hospital during the past summer. Unfortunately, the hot days caught us unprepared for as complete investigations as we should have wished. The

blood was examined in 17 cases. Among these, curious irregularities were found, making it impossible at present to do more than summarize them. In bleeding patients with sunstroke the dark color of the blood, not resembling ordinary venous blood, was noted. We had, therefore, hoped to obtain some facts regarding spectroscopic changes. Owing to the impossibility of at once obtaining the necessary apparatus a spectroscopic examination was made in only 2 cases, and in both of these the absorption band of hæmoglobin was found alone, although the appearance of the blood led us to think that possibly methæmoglobin might be present. The specific gravity was estimated in 4 cases; in 3 the specific gravity was 1055, 1056, and 1057, respectively. The fourth showed a specific gravity of 1074. This latter blood was from a patient who died two hours after admission in spite of everything that could be done for her. In this same patient the hæmoglobin value was 83 per cent., and the erythrocytes numbered 5,040,000, it being the only case in the series in which the erythrocytes were increased. The hæmoglobin was estimated in 5 other cases and amounted in these to 67, 85, 68, 74, and 68 per cent. The absence of constant high specific gravity and of increase in the number of the red cells rather surprised us, as we expected to confirm some of the results reported by others in this direction. The number of the leucocytes varied greatly. All but 3 of the severe cases examined showed at some time a high leucocyte count, but there was considerable irregularity in the time and duration of the rise. In some cases a leucocytosis of from 12,000 to 13,000 was noticed on admission. The increase was usually in the polymorphonuclears. In most of the cases in which there was a primary rise in the number of leucocytes followed by a fall and then a second increase in number, delirium tremens developed. No experiments regarding the toxicity of the serum for animals were made.

These results surprised us, especially as at the time when they were being obtained in the Ayer Laboratory of the hospital we felt justified in believing that the injection of large quantities of normal salt solution was saving some lives. To this view we still adhere.

It is a well-known fact that the treatment employed in thermic fever, viz.: cold baths, hypodermoclysis, transfusion, and bleeding, can alter materially the normal proportion of the elements of the blood.

In our cases, unfortunately, the examinations of the blood were made without any definite relation in time to the treatment as mentioned above, which might alter the findings, and in consequence vitiate somewhat the conclusions drawn, and explain in some degree the apparent discrepancies. Two explanations of the apparent contradiction of the clinical and laboratory results in our cases present themselves. It seems to us conceivable that the absence of marked elevation of the specific gravity of the blood, of its hæmoglobin contents, and of the

number of the erythrocytes may be due to the rapid passage of water from the tissues to the blood, as is seen in Hay's treatment of pleurisy with effusion, in removal of anasarca by eliminating methods, and in cholera. This theory it would be difficult to prove. A very plausible explanation of the benefit of the treatment used is that made practically certain by the work of Levene and Van Gieson as to the increased toxicity of the blood. Lambert (*Medical News*, July 24, 1897), who quotes these authors, also found that serum from the blood of some thermic fever patients caused the death of rabbits in doses of 9 c.cm. within an hour.

The serum removed at once was found by him to be less toxic to animals than was the serum of the same patients twenty to forty-four hours later.

If this auto-intoxication be the true explanation of the phenomena of thermic fever, the beneficial effects of hypodermoclysis or intravenous injection of saline solution can be readily explained by the "washing of the blood."

Treatment. The treatment of the mild cases (100° to 102° F.) can be dismissed with a few words. In this class there were 32 cases, although there are not included a large number of patients, the notes of whose condition are insufficient to allow of their classification. The indications for treatment in this class of cases were simply rest in a cool portion of the ward, the application of an ice-cap, the administration of the aromatic spirit of ammonia, or alcohol, or strychnine, and occasionally a cool bath. The cases whose temperature ranged from 102° F. to 106° F., numbered 22. These showed more severe symptoms than the milder group, and an approach to the symptoms of the higher temperatures was occasionally noted. The treatment of this class consisted in the administration of stimulants, as they were required by the state of the pulse, the application of an ice-cap, and either a cold bath or, in the more obstinate cases, the employment of rubbing with ice until the temperature approached the normal. None of these cases died. Cases with a temperature between 106° F. and 108° F. numbered 14. Three of these died, one being a man with a temperature of 107⅘° F., the second a man whose temperature was 106° F. on admission, and who later died with well-marked typhoid fever, and the third being a woman in whom alcohol undoubtedly played a more prominent part in causing death than did the result of heat. These cases were taken to the heat tent, where they were treated in the manner mentioned below in considering the cases of still more severe type, where the temperature was 108° F. or over. The cases whose temperature reached a point equalling or surpassing 108° F. numbered 22. Of these 9 died. One case of "unknown" temperature died; 1 case with "high" temperature recovered.

All of the cases except those with a temperature below 102° F. were treated in a large tent open at the ends, covered with a fly and having an asphalt graded floor. The fly was kept constantly moist by means of a lawn-sprinkler placed on the ridge-pole, an expedient that did good service and did not seem to increase the humidity. Within the tent there were two large electric fans constantly working, and faucets were conveniently arranged for the furnishing of cold water. The tent was furnished—aside from the beds—with a movable bath-tub and a medicine cabinet. In a few cases the hose was employed by directing a stream of water from a distance over the patient's body. The experience this summer in the few cases in which this method was employed rather tended to confirm the observation of former years. While this method of applying cold seemed to aid respiration it rather increased the tendency to convulsive movements. The use of the bath-tub was soon abandoned owing to the impossibility of reducing the temperature by this means with sufficient rapidity to at all keep pace with the number of admissions. In addition to this, it required more physical labor to lift the patient in and out of the tub, and to practice friction on the surface than was the case with rubbing with large pieces of ice which acted more rapidly and equally beneficially in reducing the temperature. In many cases the temperature fell rapidly to normal after rubbing with ice from ten to fifteen minutes. With the fall of temperature consciousness frequently returned, and the patient required no further attention. Frequently with the return of consciousness delirium occurred, which was at times quite violent, and in a few cases persisted for some hours. Exceptionally a second rise of temperature occurred, but seldom to such a height that it was not controlled perfectly by either a cold sponge or one cold bath.

The question as to the advisability of bleeding in cases of sunstroke has been much discussed. As a routine measure of treatment we would by *no means* advise it. In certain cases, however, where with the fall in temperature there was not a corresponding decrease in the other grave symptoms, as well as during the existence of high temperature in desperately ill cases, we found it beneficial. Feebleness of the pulse was not considered a contraindication to this procedure, and in some cases the character of the pulse improved as the blood flowed from the arm. The measure was employed in 8 cases. Of these 4 died. In regard to bleeding, as well as in regard to the two measures still to be noticed, it is to be remembered that it was only employed in the most severe cases. The mortality percentage among cases in which venesection was practised was, therefore, necessarily high. It is our belief that in no case did the removal of blood do any harm, and in many cases the abstraction of blood was followed by an improvement in the circulation, in the character of the breathing, in the color of the patient, and in

the mental state, due either to this or to the introduction of saline solution. The amount withdrawn varied from 6 to 20 ounces.

The employment of normal saline solution introduced by various channels was first advocated upon purely theoretical grounds. As has been said above, while there is some evidence that diminished serosity is present in some cases, none of the tests which we were able to employ to confirm the existence of this condition enabled us to assert its presence in our cases. This will be seen in the detailed report of certain cases, with the account of the blood picture in the cases wherein this was examined. Hypodermoclysis was employed in 5 cases. All of these were of the severest type. Of this number 1 died. It was found that this method of introducing salt solution occupied too much time to be perfectly satisfactory. We believe that valuable time may be lost in getting the saline solution into the circulation by this measure, and it has the additional disadvantage of requiring the undivided attention of at least one person during ten or fifteen minutes at a time when rapid work is essential even with a large staff of assistants. Intravenous injection of normal saline solution was found to be more applicable because of the greater rapidity with which the blood was affected, and also because of the saving of time on the part of the assistants. This measure was employed in 10 cases; of these 4 died. The operation was readily performed and took but little time, because in the large majority of cases the same incision in the median basilic vein employed for bleeding was at once used for the injection of the saline solution. Although the conditions were by no means favorable for thorough antisepsis, in spite of all efforts to accomplish this, it is to be noted that in no case did any untoward results follow from infection of the wounds made either by the lancet or by the needle.

Our conclusions in regard to the value of the last three measures are that in some cases where the other symptoms do not improve, as the temperature is reduced by rubbing with ice, the abstraction of blood from the median basilic vein to the extent of from 10 to 18 ounces causes the pulse, respiration, and color of the skin to improve. On four occasions convulsions occurred after bleeding and transfusion. While it is difficult to estimate the value of the introduction of normal saline solution, we see no evidence of its having any harmful action, and usually felt assured that the patient's condition was improved by one or the other of these procedures.

Among the sequelæ should be noted 1 case of jaundice coming on three days after onset in a case having a temperature of 110⅘° F., where there was also delirium tremens.

While from the rapidity with which these patients were brought to the hospital we were unable to make as close a study of them and to keep as careful records of their condition as we wished, we make this

report in the hope that in the future we may be able to more completely study some of the many points of interest in this condition. It seems to us that valuable results might follow the further study of the condition of the eye-grounds, the specific gravity of the blood, its spectroscopic examination, a frequent estimation of the leucocytes and a more complete study of the toxicity of the blood serum. We also believe that the introduction of normal saline solution in large quantities into the body has a beneficial result, although the few examinations of the blood which we have had made do not directly point to the method by which it causes relief to symptoms. Possibly the toxicity of the blood, a subject into which we could not enter, may explain its supposed beneficial effect.

Abstracts of Certain of the Cases.

Two of the oldest patients were aged respectively seventy-three and seventy-nine years, each of them being admitted with a temperature of 108° F. The oldest of these showed a rapid drop in temperature with improvement in all of his symptoms under rubbing with ice. The younger one, aged seventy-three years, did not recover so completely on rubbing, and was given a pint and a half of normal salt solution under each breast, with rapid improvement of all of his symptoms. In the older man, aged seventy-nine years, the temperature did not reach the normal point until four days after admission. Another patient aged seventy-five years, was admitted with a temperature of 107⅜° F., and rapidly improved under treatment. He has since responded to our inquiry regarding his health at present, that he is weak and unable to walk—possibly as much from the effect of years as from the effect of heat.

Case 1025 should be included in the list of sunstroke occurring in old people. He was a man, aged seventy years, with a temperature of 110¼° F. on admission, and with all the signs of senile degeneration well marked. Notwithstanding the vigorous application of cold his temperature remained high, rising to 108° F. after the primary fall to about 103° F. He died on the fourth day with a continued high temperature. He was neither bled nor transfused.

Case 1258.—P. M., male, aged forty-one years; brought in with a temperature of 110⅗° F. He was bled 12 fluidounces, and transfused 1 quart. His temperature fell to 101⅖° F. in about four hours, and he regained consciousness. The next day all he complained of was a slight headache. His knee-jerks were absent. He was discharged the next day feeling perfectly well. On the day of admission, hour not mentioned, his leucocyte count was 7500, and when discharged the count was also 7500.

Case 1138.—W. W., male, aged thirty-eight years; a moderate drinker of beer and ale, was admitted with a temperature of 106⅗° F., conscious, and with a diffused red rash over the body; he was given an ice-rub, and in two hours his temperature had fallen to normal, and

with the exception of a headache he felt well. He was discharged the next day. His blood examination on admission showed leucocytes, 9200, and on the following day 9850.

Case 914.—H. C., male, aged fifty-two years; admitted June 23, 1901, with a temperature of 104° F., conscious, although somewhat stupid and with severe headache and backache. His pupils were equal and contracted. He was given ice-rubbing and bathing. His temperature did not fall until the eleventh day, having been kept up by an intercurrent attack of enteritis. A leucocyte count taken on the day of admission, immediately after the ice-rub, gave 16,000. On the following day the specific gravity of his blood was 1055, the hæmoglobin 68 per cent., and the erythrocytes 4,906,000.

Case 1025.—J. W., male, aged seventy years; brought into the hospital in an unconscious condition, with a temperature of 110⅘° F., pulse feeble, pupils contracted, face cyanosed, head retracted. He was given an ice-rub, which was continued for over an hour, by which time the temperature had fallen to 102° F., and he became partially conscious, although unable to speak or understand. His arteries were very atheromatous, and arcus senilis was present. History of the previous free use of alcohol was not obtained. Knee-jerks were entirely absent. The urine was acid, with specific gravity of 1019. There was a ring of albumin, but no sugar. A few granular casts were found. After admission to the wards his temperature rapidly rose again, and on the fourth day he died despite the application of ice and the use of strychnine and nitroglycerin. The thermic fever in this case was merely the last chapter in a case of an old man with general arteriosclerosis. Before the ice-rub his blood was examined with the following result: specific gravity, 1056; erythrocytes, 4,225,000; leucocytes, 13,500; hæmoglobin, 85 per cent.

On July 2d, the next day, 10.30 A.M., hæmoglobin, 75 per cent.; leucocytes, 9850.

On July 3d, time not given: leucocytes, 11,000. Differential count: polymorphonuclears, 94.8 per cent.; small mononuclears, 48 per cent.; large mononuclears, 4 per cent.; eosinophiles, none.

Case 1044.—L. R., female, aged twenty-seven years; brought into the hospital unconscious, and with a temperature of 110° F. Her temperature fell to 105° F. under ice rubbing in three-quarters of an hour; she then had two convulsions, which were relieved by one-quarter of a grain of morphine hypodermically. Later in the day she was transfused with normal salt solution. For hours she was in constant tetanic spasm, and at times was quite noisy. Her temperature subsequently remained above 105° F. She had constant ice-rubbing, with no avail. She died twelve hours after admission.

Case 1126.—C. Z., male, aged thirty-nine years; was brought into the hospital with a temperature of 110° F., unconscious and pulseless, with cyanosed face, contracted pupils, and retracted head. He was rubbed with ice, and bled 11 fluidounces, and then transfused with one and one-half quarts of normal salt solution. Strychnine and digitalis were given hypodermically, and one pint of cold water enteroclysis given. When his temperature fell to 105° F. he went into violent convulsions. Two hours after admission his temperature had fallen to 102⅘° F., and he regained consciousness, but by evening his temperature had risen to 106° F., and he was again unconscious. The next day consciousness

returned and he developed delirium tremens, and it was then learned from his friends that he was seldom sober. On the third day his temperature rose to 106° F., and he died with signs of pulmonary œdema. On the day of his death, the third day after admission, his blood gave the spectrum of dilute hæmoglobin only.

Case 1130.—J. B., male, aged forty-nine years; was brought in unconscious with a temperature of 110° F., with rapid pulse, widely dilated pupils, and retraction of the head. This patient responded so rapidly to the ice rub that neither hypodermoclysis nor transfusion was resorted to, the temperature falling to normal in three hours. His knee-jerks were absent.

Case 1129.—S. R., male, aged sixty-three years; was admitted unconscious, with a temperature of 106° F. and pulse 132; he had incontinence of feces, and was very restless and noisy. He was given an ice-water tub, but the temperature only fell one degree, when a profuse rash appeared. After an ice-rub the temperature fell to 102⅘° F., and two hours after admission it was nearly normal. The man subsequently developed delirium tremens. His urine contained no albumin, but did contain a few granular casts. On the fifth day his leucocytes were 12,125. He made a perfect recovery, and was discharged on the twelfth day.

Case 1141.—W. J., male, aged fifty-seven years; a heavy drinker, was brought in with a temperature of 109⅗° F., profoundly unconscious, with strong odor of alcohol on his breath. He was rubbed with ice, and bled 10 fluidounces and transfused one quart of normal salt solution. A hypodermic injection of strychnine ($\frac{1}{30}$ grain) was given. In three hours the temperature fell to 99⅘° F., he then went into severe convulsions, which were quieted by a hypodermic injection of ¼ grain of morphine. On the third day his temperature began to rise, and he went into delirium tremens, and after developing Cheyne-Stokes respiration died on the following day. The leucocyte count was made immediately after admission, during transfusion, and showed 13,200. On July 4th, the day after admission, the count was 9200, and on the 5th, while in delirium tremens, the leucocyte count was 14,650. His blood gave the spectrum for hæmoglobin only.

Case 1161.—S. B., male, aged forty-one years, colored; brought in unconscious with a temperature of 108° F. An extremely muscular man, weighing 195 pounds. He was given an ice-pack, was bled 8 fluidounces, and transfused one quart. In less than two hours his temperature had fallen to 99° F., he then went into severe tetanic convulsions, for which morphine was given. By evening he regained consciousness, and felt none the worse for his experience. His knee-jerks were absent. He was discharged feeling well in two days. When seen ten months later his only complaint was an absence of sexual power.

Case 1142.—M. K., male, aged fifty-three years; was admitted at 2.15 P.M. on July 3d, with a temperature of 108° F. He was not unconscious, but was very weak, his pulse being not perceptible, but after the administration of ammonia it became so, and was 140 per minute. Under the ice-rub his temperature rapidly fell and became normal in less than two hours. He was discharged the next day apparently well. His leucocyte count before the ice-rub was 12,160, but by the next day it had fallen to 8700.

Case 1014.—L. N., female, aged twenty-one years, was a large healthy-looking woman; she was conscious when admitted, and with a temperature of 107° F. She had been ailing for some days before admission, and had had a convulsive seizure of some kind on the night before. She was rubbed with ice and given ammonia, but her temperature did not fall to normal until the next morning. She was discharged well on the third day. Before the ice-rub the specific gravity of her blood was 1057. The other blood examination was made after the ice-rubbing, and was as follows: Hæmoglobin, 65 per cent.; erythrocytes, 4,166,000; leucocytes, 10,250. On the next day the leucocytes were 7350, and on the day of discharge 8860.

Case 1027.—E. G., male, aged forty-eight years, tailor. Patient was overcome in his shop and was brought into the hospital in an unconscious condition, having but recently recovered from a debauch. His pulse was rapid, hard, and tense, his temperature was 110° F., his face cyanosed, his body rather rigid, and his head retracted. Patient had involuntary fecal movements. He was rubbed with ice, but as the temperature did not seem to fall quickly under the icing, a vein was opened in the arm and the patient bled 6 fluidounces. Two quarts of normal salt solution were given into the opened vein, and in about one-half an hour his temperature fell to 104½° F. He then went into a general convulsion, which, however, affected the hands especially. Five hours after admission the patient was still unconscious, with stertorous respiration, but with a better pulse. Under further treatment with ice his temperature fell to 102° F., but by evening, after admission into the ward, it rose to 105° F.; tub-bathing was resorted to, but it was not until next morning that he regained consciousness. The urine then showed: specific gravity, 1019; ring of albumin, no sugar, dark granular casts containing blood. His knee-jerks were entirely absent. There was no paralysis. He remained in the hospital eleven days, and was discharged well but weak. When seen ten months later he expressed himself as feeling perfectly well, with the exception of an entire loss of sexual power, a condition which had not existed before. His urine was normal. Immediately after the ice-rub his blood was examined and showed: hæmoglobin, 67 per cent.; erythrocytes, 4,352,000; leucocytes, 8500. The leucocyte counts in this case subsequently were as follows: July 2d, 12.30 P.M., 10,466; 3d, 10.30 A.M., 10,250; 5th, 6 P.M., 8160; 8th, 10 A.M., 6000.

Case 1039.—E. G., female, aged fifty-eight years; brought into hospital unconscious with a temperature of 108° F., with a full, strong pulse, and cyanosed face. A vein was opened in the arm, but before anything else could be done she died, fifteen minutes after admission.

Case 1033.—Aged seventy-nine years, was admitted with a temperature of 108° F., and recovered promptly under treatment. Twelve minutes after admission the leucocytes were counted and found to number 11,750; polymorphonuclears, 72.4 per cent.; small mononuclears, 23.2 per cent; large mononuclears, 4.4 per cent. On the day after admission, without evident reason, the leucocytes numbered 15,000. On the third day after admission they numbered 6000; on the day of discharge, six days after admission, they were 8000.

Case 1038.—Was admitted at 4 P.M., on July 2d, with a temperature of 110⅘° F. On admission the leucocytes numbered 14,800; on July 3d, 11,320; on July 5th, 7850. He was discharged cured July 5th.

Case 1123.—Aged seventy-three years, was admitted on July 2d, with a temperature of 108° F. Beside rubbing with ice he was given one and one-half pints of normal saline solution under the skin. On the day of admission the leucocytes numbered 8525. July 3d, without evident cause, they numbered 9730, on July 5th, 13,800. He was discharged cured on the latter date.

Case 1034 was almost moribund when he was admitted at 2.30 P.M. on the afternoon of July 2d. Unconsciousness was complete; respirations were shallow and almost imperceptible, the pulse could not be felt at the wrist, the face was cyanosed, and there was present the curious mixed tonic and clonic convulsion so characteristic of thermic fever. The temperature in the rectum was 112° F. For a few minutes artificial respiration was practised, and free stimulation was instituted from the beginning. After a few minutes of artificial respiration oxygen was employed with benefit, while the patient was being rubbed with ice. By means of ice the temperature was shortly reduced to 104⅘° F., but as the patient's cyanosis, complete unconsciousness, and extremely feeble cardiac action with shallow respirations continued, he was bled to the extent of 16 ounces. Bleeding improved the character of the pulse, but the other symptoms still remained in evidence. A pint of saline solution was introduced under the skin and another pint was injected into the median basilic vein. The respirations became deeper and the pulse stronger, while the temperature fell to 100⅗° F. It was hoped that he would recover in spite of the fact that the convulsive movements continued after the above measures had been adopted, but at 8.30 P.M. on the same evening the patient suddenly died, his temperature having again risen to 105⅕° F. not long before death.

Case 1037.—An Italian, aged thirty-five years, was in a dangerous condition when admitted to the heat tent at 3 P.M. on July 2d. His temperature on admission was 109⅗° F. He was completely unconscious, strongly convulsed, with cyanotic face, and passing involuntarily the characteristic yellowish liquid stools. Rubbing with ice soon brought the temperature down to 103⅘° F., but unconsciousness and convulsions and cyanosis continuing, ten ounces of blood were withdrawn from the arm, a pint of saline solution was introduced into the cut vein, and one and one-half pints were given by hypodermoclysis. The notes state that before treatment the pulse was imperceptible and the respirations had almost ceased. After the bleeding and salt solution injections the condition steadily improved, and at 6.30 P.M. the patient was conscious, sat up in bed and asked for a drink of water. In this case the temperature did not reach the absolute normal until four days after his admission, although there was no evidence of any reason for its continued elevation. Before being given his ice-bath the blood was examined as regards the number of leucocytes. They were found to number 8000; polymorphonuclears, 89.2 per cent.; small mononuclears, 9.6 per cent.; large mononuclears, 1.2 per cent. On the next day the leucocytes numbered 13,225. On July 5th (two days after the last count) they numbered 6350. Spectroscopic examination showed only the absorption band of hæmoglobin.

Case 1047.—This case showed several interesting features. When admitted, at 4 P.M. on July 2d, he was unconscious, with stertorous breathing, cyanosis of the face, clonic convulsions, and a temperature of 110⅗° F. He was vigorously rubbed with ice, and during this pro-

ceeding struggled violently, although still entirely unconscious. The rubbing quite rapidly reduced the temperature, but the other chief symptoms still continued, and 20 ounces of blood were removed from the median basilic vein, followed by the injection of a pint of normal saline solution, and the injection beneath each pectoral region of one pint of the same material. After the primary fall his temperature rose again to 106⅕° F., between 7 and 8 o'clock in the evening, but speedily fell on a repetition of the rubbing with ice. On the next day he was perfectly rational and conscious, but showed marked icterus of the skin and conjunctiva. Four days after admission a typical attack of delirium tremens began and continued for three days. In spite of the deep pigmentation of the skin no bile was present in the urine. At no time could enlargement of the liver or gall-bladder be detected, and there was no evidence of pain, although this feature might have been concealed by his delirium. The jaundice persisted until his discharge, in apparently perfect health, on July 15th, thirteen days after admission. His temperature was irregular until July 12th, when it remained at about the normal point. On the day of development of the delirium tremens his leucocytes numbered 7500. Two days later the number rose to 9640.

Case 1190.—This case, dying with a temperature of 106° F., cannot be wholly attributed to thermic fever. While it cannot be denied that many of her symptoms and probably the exact time of her death were determined by the effect of heat, she died as does a case of alcoholism, not at all as does one of thermic fever. Death, therefore, should not be attributed to the latter cause.

Case 1007 was a large, fat Italian, admitted with a temperature of 108° F. He was semiconscious and delirious. The pupils were dilated, and there was combined horizontal and vertical nystagmus. In spite of his high temperature his pulse had a fair volume, which was very unusual in cases reaching that degree of severity. After a bath in iced water and being rubbed with ice, there was noticed what has been frequently seen in these cases, a profuse papulo-vesicular eruption of a dull-red color over the chest and abdomen. The temperature fell to 103⅕° F. after treatment for half an hour, and at the same time the delirium was replaced by perfect consciousness and a rational state of mind. The urine examined on the day after admission showed a trace of albumin with a few hyaline and granular casts. The notes state that the reaction for sugar was present, but no statement is made in regard to the test employed. In this case the temperature did not reach normal until one week after admission, although there were no new signs or symptoms explaining this continuous elevation. On the day before his discharge, one week after his admission, the albumin was still present (0.3 per cent.), but no sugar was found. Immediately after his first bath the blood was examined and showed: erythrocytes, 4,720,000; leucocytes, 16,000; hæmoglobin, 75 per cent. Nine hours later (June 30th): erythrocytes, 4,700,000; leucocytes, 9000; hæmoglobin, 74 per cent. Next day (July 1st): hæmoglobin, 73 per cent.; leucocytes before ice-sponge, 9800; leucocytes after ice-sponge, 7228. July 2d: leucocytes, 9600; 3d, 7250; 5th, 8300.

Case 1013.—A woman, aged thirty-six years, had her blood examined ten minutes after her first iced bath. The specific gravity was 1074; hæmoglobin, 83 per cent.; erythrocytes, 5,040,000; leucocytes, 12,500. Death occurred about two hours after the above count was made.

Case 1022.—A baby, aged thirteen months, was admitted with a temperature of 107⅘° F. It died before anything could be done for it, five minutes after admission. From the notes it is not possible to say definitely whether or not this was a case of intestinal intoxication.

Case 1023.—Admitted with a temperature of 111° F., died within eleven minutes of his admission to the hospital before any energetic treatment could be adopted.

Case 1024.—Admitted with a temperature of 111⅘° F., died within half an hour of admission, never recovering consciousness.

Case 1048.—Was dying when admitted, with a temperature of 111° F. Death actually occurred two minutes after admission to the hospital.

CHRONIC EMPYEMA OF THE FRONTAL, ETHMOIDAL, AND BOTH SPHENOIDAL SINUSES;

WITH EXTENSIVE NECROSIS, COMPLICATED WITH ADENOMA OF THE POSTERIOR ETHMOIDAL AND SPHENOIDAL REGIONS.*

By Joseph H. Bryan, M.D.,
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The bone necrosis frequently associated with suppurative inflammation of the accessory sinuses is, in the earlier stages, so often overlooked by the practitioner that the following interesting record of failure and success is of much interest:

Mrs. ——, aged forty-eight years, consulted me in the latter part of January, 1899, complaining of excruciating headaches, referred to the left side of the head, but with greatest intensity over the left frontal region. The pain was constant and has been increasing in severity until now it has become unbearable. She attributes the beginning of the headaches to a period about eight years ago, when during the convalescence from an attack of pneumonia she was seized with a most violent pain in the head, which was accompanied by great prostration. The condition at the time was described by her physician as a congestion of the brain. The pain passed off, however, within forty-eight hours. From that time until the present she has been annoyed by the dropping of mucus into the nasal space. This has increased within the past two years; and recently the discharge has become very fetid, so much so that she suffers continually from morning sickness. With the increase in the discharge the headaches have also increased, but whenever there is a diminution of the flow of the secretion the headaches become intensified. On examination there was no swelling about the face; but there was great pain on firm pressure over the supra-orbital ridge, and very great sensitiveness on pressure just under the ridge near its inner angle.

A rhinoscopical examination disclosed only a moderate amount of swelling of the middle and inferior turbinals on the left side, and a

* Read at the Thirty-third Annual Meeting of the American Laryngological Association, held in New Haven, Conn., on May 27, 28, and 29, 1901.

very slight deviation of the septum to the right. The middle meatus was to a considerable degree obliterated by the overhanging middle turbinal. No secretion was observed in the anterior nasal chamber. A posterior rhinoscopical examination showed the posterior extremities of the inferior and middle turbinals on both sides to be normal in color and size. Some thick, yellowish secretion was observed flowing into the postnasal space over the posterior extremity of the left middle turbinal. After thoroughly cocainizing the parts, the probe revealed extensive caries of the anterior ethmoid region. Examination of the frontal and maxillary sinuses with the electric light was of negative result.

The serious character of the patient's condition was explained to her; that there was an abscess of the frontal and ethmoidal sinuses with extensive bone disease, and an operation was advised; but upon her earnest solicitation an external operation was to be postponed until all intranasal methods had been tried.

April 22, 1899. Complaining of pain in the left eye, a blurring sensation, and with a feeling that the left eye is larger and heavier than the right. Examination by Dr. Wilmer, who had examined her eyes in 1896, found the refraction the same as at the first examination.

The curvature of the cornea in the left eye slightly changed, the slight astigmatism with the rule being less than in 1896. Vision in the left eye 20/20 as before, but now print near at hand seems clearer with the right eye than with the left. In both eyes fields for white and other colors are normal. Fundus normal, no pulsation of retinal vessels. The anterior ethmoidal cells were frequently curetted and much diseased bone was removed from time to time, giving the patient temporary relief by a freer drainage; this improvement, however, was only of slight duration.

May 10*th*. An unusually large piece of bone was removed from the anterior ethmoidal region by the curette, thus admitting of the passage of a canula into the frontal sinus, which was washed out with a saturated solution of boric acid; a large quantity of thick, fetid pus was brought away. This was repeated on several different occasions, but instead of giving the patient relief only increased her suffering to a very great degree. The passage of the canula into the frontal sinus revealed such an extensive disease of the bone that all hope of removing it through the nose was abandoned; and the pains increased to such an extent that the patient readily consented to more radical measures being taken for her relief.

June 4*th*. The patient having been prepared for the operation, and the left eyebrow shaved, she was placed under ether anæsthesia. An incision commencing just within the supra-orbital notch was made over the supra-orbital ridge down to the bone, extending as far as the nasal eminence. The flap thus formed, composed of skin and periosteum, was elevated and a button of bone about 1 centimetre in diameter was removed from the anterior wall of the sinus at a point about one or two lines from the median line and just above the supra-orbital ridge.

Upon opening the sinus it was found filled with thick, fetid pus and granulation tissue. After the removal of the contents of this cavity, the probe revealed extensive disease of its bony walls; there was some softening of its posterior wall near the floor, the partition between the two sinuses was extensively diseased and broken down, and the roof of the orbit was in such a necrotic state that a piece about the size of a

dime had to be removed. After the removal of all the diseased bone within the sinus by means of the curette, the ethmoid bone was next examined and found extensively diseased in its anterior region. With the little finger within the nose as a guide, the curette was passed from the sinus into the nasal chamber and all diseased bone that could be felt was removed, thus leaving a very large communication between the sinus and the nose. The sinus was then irrigated with a solution of bichloride of mercury 1 : 3000, its walls touched with a 20 per cent. solution of chloride of zinc and packed with iodoform gauze, one end of which was brought down through the nose to act as a drain. The wound was then closed, the edges of the periosteum being drawn together with catgut, while the external wound was closed with silk sutures, and the whole hermetically sealed with iodoform and collodion.

At the end of the second day the gauze drain was withdrawn through the nose, and through a canula passed through the nose into the sinus it was irrigated with a saturated solution of boric acid. The secretions rapidly subsided under daily irrigations, and in the course of three weeks they had practically ceased. The stitches were removed on the sixth day, perfect union having been obtained.

The patient was completely relieved of her headaches, her convalescence was rapid, and she expressed herself as feeling better than she had felt for several years. At the end of a month from the time of the operation she had sufficiently recovered to leave for York Harbor, where she was to spend the summer. At this time there was no headache and no secretion coming away when the sinus was irrigated. A rhinoscopical examination showed the nose free from secretion and in otherwise good condition, save for an enlargement of the anterior extremity of the middle turbinal, which had not been removed.

July 15th. She contracted a severe head-cold, and in the course of twenty-four hours the skin over the left frontal sinus was very red and swollen, the swelling involving the upper and lower lids of both eyes. In consequence of no secretion passing into the nose, the pain became excruciating; and as immediate relief was demanded, an opening was made into the sinus by Dr. Sprigg, her family physician, at the seat of the greatest swelling, which was some distance above the supra-orbital ridge, evacuating a large quantity of pus, thus affording considerable relief. The following day she was removed to Boston to be under the care of Dr. Coolidge, to whom I had previously referred her in case of necessity during my absence abroad. The suppuration being so profuse, and the passage between the nose and the sinus having closed, thereby preventing drainage through the nose, Dr. Coolidge enlarged the external opening, at the same time making a very free communication between the sinus and the nose. The wound was not closed, but treated by the open method. Although the patient was relieved to some extent of the pain, the suppuration never ceased. When she returned to Washington in October her condition was as follows:

There was an opening into the sinus about half an inch above the supra-orbital ridge and a small fistulous tract below this ridge just above the internal canthal ligament, through which pus came in large quantities; there was also a free discharge of pus through the upper wound, but little or none passing into the nose.

A probe passed into the sinus through the external wound showed extensive necrosis of the floor of the cavity, especially that portion in

relation with the ethmoid cells. It was found impossible to pass a probe from the nose into the sinus, the opening previously made by myself and Dr. Coolidge having closed completely.

The open method of treating the sinus was continued, the cavity being irrigated daily with one or more of the several antiseptic solutions employed, such as hydrogen dioxide, formalin, or a saturated solution of boric acid. In addition a gauze wick was introduced through the external opening to facilitate drainage. This method of treatment was faithfully followed for several months, with the expectation of my being able to re-establish the opening into the nose by means of the curette; but failing in this and in view of the tendency to the closure of the external opening, the patient's condition in the meantime growing worse, it was decided to open the sinus again.

January 7th, 1900. Having been prepared for the operation, the eyebrow shaved, she was etherized, an incision was made through the skin and periosteum in the line of the incision over the supra-orbital ridge in the original operation, the flap elevated, and the sinus exposed. The upper half of the cavity was filled with firm granulation tissue, while on the most dependent part of the floor pus was found in considerable quantity. After thoroughly cleaning the cavity out, the probe revealed extensive necrosis of the ethmoid bone, as well as that portion of the floor of the sinus in relation with it. As much of the diseased ethmoid bone as could be reached by means of a curette passed through the frontal sinus opening, using the little finger within the nose for a guide, was removed. The probe passed not only far back on the affected side, but it could be passed diagonally through to the opposite side without detection of any further diseased bone. It was thought that the probe must have reached the sphenoidal sinuses, although there was no positive means of verifying the belief. After thoroughly irrigating the parts with a solution of bichloride of mercury 1:3000, and packing the cavity with iodoform gauze, one end of which was drawn out through the nose, the wound was closed as in the original operation.

The night of the operation and the day following, the patient was very ill, complaining of intense pain on top of the head, and in the occipital region. During the night and part of the following day she was delirious. There was only a slight elevation of the temperature.

9th. Mind much clearer, pain in top of the head less, but she still complains of intense pain in the occipital region. The gauze drain was removed and the frontal sinus irrigated with a saturated solution of boric acid through a canula passed through the nose into the cavity. This was accompanied by a great deal of pain in the frontal region, but no pus was observed in the return flow. The sinus was irrigated daily, but with some difficulty, owing to the pressure of the middle turbinal, which interfered with the passage of the canula; although the anterior extremity of this bone had been removed by Dr. Coolidge and several pieces by myself, prior to this last operation, it still acted as an obstruction. The stitches were removed on the sixth day, good union having been obtained, and firm closure of the fistulous openings above and below the supra-orbital ridge. The patient expressed herself as feeling greatly relieved, all the occipital and frontal pains had disappeared and she seemed to be making a rapid and satisfactory convalescence, although the probe passed into the region of the anterior

ethmoid cells still revealed some necrosed bone; there were no sections found in the anterior nasal chamber nor in the postnasal space, and nothing abnormal was observed by posterior rhinscopy.

February 10th. One month after the operation, when everything seemed to be progressing favorably, she was seized with a violent headache referred to the frontal and temporal regions. This was accompanied by obstinate nausea and vomiting. The temperature, which up to this time had been normal, went up to 100° F. On attempting to wash out the frontal sinus the parts had become so swollen it was found impossible to pass the cannula. On using the probe under the middle turbinal an over-distended ethmoid cell was evidently penetrated, for a considerable quantity of foul-smelling pus made its appearance in the region of the middle meatus on its withdrawal. This gave her some relief, for the violence of the pain subsided, but there was a constant dull headache remaining. She complained of an intense itching of the skin over the left frontal region, which at times became very distressing. This sensation seemed always to be associated with some new invasion of the ethmoid cells.

From this time my efforts were directed to the removal of the diseased ethmoid bone; first removing portions of the middle turbinate, then with the curette and cutting forceps, diseased bone was removed as fast as was compatible with safety. The headaches continued with greater or less severity until about the early part of May, when they increased in intensity, the pain being referred principally to the supra-orbital and infra-orbital regions, and in the left eye. As the disease was evidently progressive in character she was put on a course of the iodide of potash, commencing with ten drops of a saturated solution three times a day; it was carried up to the point of tolerance, which was forty drops three times a day, but without the slightest benefit resulting.

June 1st. Pain in the left eye much worse than it has previously been, photophobia, lacrymation, and congestion of the conjunctiva, apparently no deviation of the eyeball from its normal axis. Palpation shows a swelling along the internal portion of the floor of the orbit, extending upward to the internal angle, and very painful to the touch. Ophthalmoscopical examination by Dr. Wilmer showed a pulsation of the ophthalmic veins.

As a result of the consultation it was thought that the ethmoid cells in relation to the inner wall of the orbit were involved, and in order to prevent an orbital abscess an operation was decided to be immediately necessary. The eyebrow was shaved and on the following day, June 2d, she was put under ether anæsthesia. An incision commencing at the juncture of the middle and outer third of the supra-orbital ridge was made through the skin and periosteum along the ridge to its internal angle, then curved and carried along the inner border of the nose to within an inch of its tip; the periosteum elevated above and below, thus exposing the contents of the orbit. There was found to be a bony swelling along the inner part of the floor and the inner wall of the orbit, extending some distance backward. The bone was so thin and soft that the weight of a light perforator passed between the trochlea and internal canthal ligament was sufficient to penetrate it, showing that the lateral wall of the orbit about the region of the os planum had become so thin that a little longer delay would probably have resulted in an orbital abscess. A sharp curette was passed through this open-

ing, bringing away some pus and a large quantity of soft and friable bone tissue. All diseased bone that could be detected was removed in this way. It was thought best to explore the frontal sinus; accordingly, the periosteum was stripped up sufficiently to expose that cavity thoroughly; it was, however, found filled with firm granulation tissue. Owing to the possibility of infection from the lower wound, the sinus was with some difficulty cleared out; after cleansing the parts a strip of iodoform gauze was passed through the ethmoidal opening into the nose, the periosteal wound closed with catgut and the external wound with silk sutures, the whole sealed with iodoform and collodion and protected by a bandage.

3*d*. Complains of little or no pain this morning, but has been more nauseated than after the previous operations; temperature 99° F.; considerable swelling of the upper eyelid and face, otherwise doing well.

4*th*. No headache, temperature normal, swelling of the upper lid very much less; removed gauze from the nose and irrigated with a saturated solution of boric acid; complains of intense vertigo and nausea.

5*th*. Swelling of the face and upper eyelid very much diminished; vertigo increasing; no pain; temperature normal; upon irrigating the nose only small particles of secretion brought away.

6*th*. Vertigo still very marked, swelling sufficiently subsided to admit of a thorough inspection of the eye, which was found to be in good condition; complains of photophobia and double vision. These uncomfortable and annoying symptoms continued for several weeks, gradually passing off. Eye kept bandaged to exclude the light. On the sixth day the stitches were removed, the wound having thoroughly healed.

12*th*. Complained this morning of feeling chilly; temperature, 99° F.; evening temperature, 102° F. Examination of the wound shows no swelling nor soreness, but there is some swelling of the middle ethmoid region; no secretions have been brought away by the irrigations.

13*th*. No further chilly sensations; feeling better; temperature 8 A.M., 100° F.; 12 M., 100° F.; 4 P.M., 99° F.; 8 P.M., 99° F. Some pus was brought down by irrigation.

14*th*. Vertigo and diplopia still continue, although to a less degree; secretions coming away freely; morning temperature normal, evening temperature, 99° F. No headache.

20*th*. Decided improvement in the patient's general condition since the last note; vertigo slight; diplopia has disappeared, no photophobia, no pain in or around the eye, which has returned to its normal appearance. Secretions, mucopurulent in character, but much less in quantity, coming from the middle ethmoid region.

Having improved sufficiently, she was removed to Chevy Chase, a suburb of Washington, for the summer. For the first ten days of her stay in the country her condition continued to improve, but on or about July 1st she contracted a severe attack of membranous colitis, which greatly prostrated her. During the acute illness, which lasted during the month of July and part of August, little or nothing could be done in a surgical way for the relief of her local trouble. The nose was irrigated daily with a boric acid solution, by which means a free

drainage was maintained. Up to the time of her removal to the country the middle turbinal and the ethmoid cells as far back as the middle of that bone had been completely removed. Although the probe still revealed caries of the ethmoid cells, and the secretions while greatly diminished in quantity still contained pus, the disease did not seem to be active. She was freer from pain in the head than at any time since the relapse in February.

September 12th. To-day she had a violent pressure headache; the secretions have practically ceased; refers the greatest pain to the occipital region and to the vertex; pressure back of both eyes, but most severe on the left side. A decided swelling is observed for the first time over the left eye near the inner angle of the orbit. Unfortunately, no ophthalmoscopical examination was made at this time. Upon examination of the ethmoid bone with a probe a small sinus was discovered, which extended some distance backward. This was enlarged with the curette, which

FIG. 1.

Knife for opening sphenoidal sinus.

permitted a free flow of secretion with much relief to the patient. The headaches now became quite frequent and were referred principally to the occipital region.

From this time on the remaining middle turbinal and the ethmoid bone were attacked with the cutting forceps and curette.

October 5th. Quite a large bony growth was observed for the first time, triangular in shape, with its base attached to the middle meatus, extending across the vestibule of the nose. After the removal of this bony obstruction a firm growth, grayish in color, was discovered springing from the ethmoid bone between the middle and superior turbinals and in the region of the posterior ethmoid cells; it was quite firm in consistency and did not bleed readily when touched with the probe. This growth blocked up the upper portion of the nose, pressing firmly against the posterior portion of the vomer, destroying the nutrition of this part of the septum and causing necrosis of the bone. After a partial removal it was found to extend backward and to be attached to the anterior wall of the left sphenoidal sinus.

This cavity was now opened by means of a bony flap being moved from its anterior wall by a peculiar shaped knife made for me by Tiemann & Co., shown in Fig. 1.

This knife is sharpened on two sides, the lower, which can penetrate the cavity, and the anterior with which a forward movement can be made, cutting through the anterior wall, then reintroducing it into the cavity at a different angle, a bony flap can be formed, the removal of which leaves quite a large opening in the sinus. After opening the sinus in the above manner it was found filled with thick, foul-smelling pus, the evacuation of which gave the patient the greatest relief. The occipital headaches, which had been almost constant, now ceased for a while.

After the left sphenoidal sinus was exposed, the probe revealed a very extensively diseased condition of the sphenoid bone; the septum between the two sinuses was carious and partly broken down, so that the two cavities communicated and were involved in the suppurative process; the roof of the cavity, at its junction with the nasal septum, as well as its floor, were also in a necrotic state. From time to time this diseased bone has been removed by means of the cutting forceps, giving the patient only temporary relief, for as fast as the growth is removed it forms again, blocking up the opening into the left sphenoidal sinus, which is used to drain both cavities. When the drainage is interfered with the pressure at the back of the eyes becomes very severe, and the headaches, which are referred to the occiput, vertex, and frontal regions, become almost unbearable. It has been necessary to cut away the growth about once in ten days or two weeks, but now, as it is growing with greater rapidity, it is necessary to operate oftener.

At the present writing of this paper the condition of the patient is as follows:

Both sphenoidal sinuses are the seat of the suppurative process, nearly the whole of the ethmoid cells on the left side having been removed; the anterior wall of the left cavity, part of the roof and floor, and the partition between the sinuses have been removed; yet in spite of the extensive bone removal, it is impossible to keep the cavities drained, owing to the location and rapid development of the growth. The indications at present are that it is extending into the deeper parts of the sphenoid bone, while the patient's strength is rapidly failing and her suffering greatly increasing, and it does not seem possible that any but an unfavorable termination of this unfortunate case is to be looked for.

The following is a report of the bacteriological examinations made by Dr. William Gray, of the Army Medical Museum:

On two occasions inoculations from the secretions of the sphenoidal cavities of Mrs. —— were made. Each time six agar tubes were used.

Of the first batch of tubes all showed a growth within twenty hours; the growths consisted of staphylococci pyogenes aureus and albus; the albus predominating. The growth in the tubes was not excessive, several showing the presence of but two or three colonies, while the tubes that contained the largest number of bacteria did not develop more than eight or ten colonies. It was thought at the time that besides the staphylococci there was a diplococcus present, but after repeated transfers this organism also proved to be a staphylococcus.

Of the second batch of six tubes, but three developed any growth, the other three remaining sterile. The growth proved to be the same as in the first lot of tubes, staphylococci.

Toubert,[1] in a paper on the intracranial complications following disease of the sphenoidal sinus, gives full details of two cases which came under his personal observation, together with a summary of twenty-two

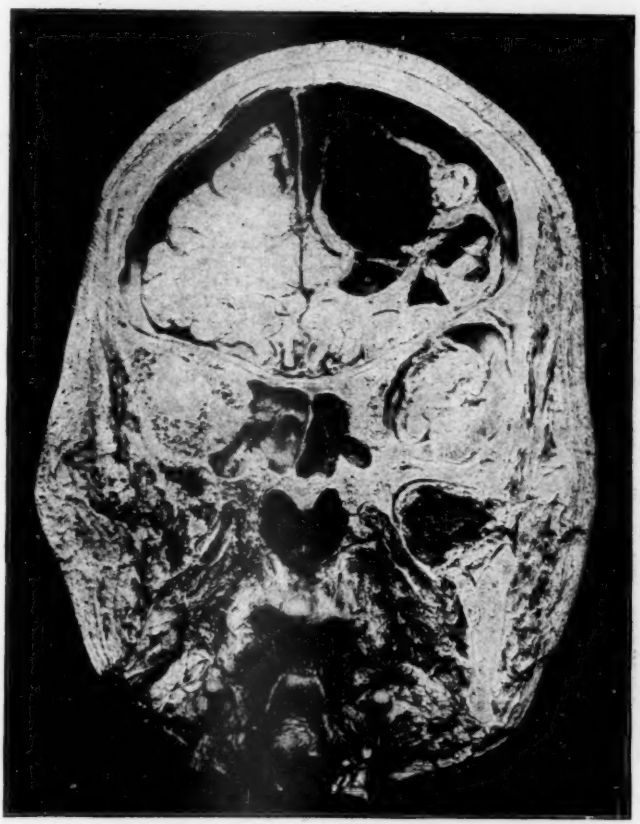
FIG. 2.

A photograph of two dry preparations, the upper picture showing a very large right sphenoidal sinus with its foramen located just below the posterior extremity of the superior turbinal, while the opposite half of the sections shows a comparatively small left sphenoidal sinus. The lower picture shows two complete left sphenoidal cavities with one foramen (B) in the lower.

others collected from various sources. He attributes the exciting cause to infection, sometimes by the bacillus of influenza, but principally by the pneumococcus or diplococcus of pneumonia, which occur both in the pus of the sinus and in that of the meninges, the organisms having made their way to the sphenoidal sinus from the nose, the nasopharynx, or from another sinus, as, for example, the ethmoid.

Infection may take place directly in connection with a general disease, such as pneumonia or erysipelas, or indirectly, (1) after febrile diseases, such as typhoid fever and influenza, or (2) after chronic diseases, such as syphilis, which tend to produce necrosis of the bone.

FIG. 3.



A photograph of a section made through the posterior third of the sphenoidal cavities, showing the foramina, A A, located about the middle of the anterior wall. The right sinus is well developed, while the left is partially subdivided by an incomplete partition. An additional point of interest in this specimen is a large abscess sac, which occupied nearly the whole of the frontal lobe, and a second sac occupying part of the temporo-sphenoidal lobe.

It is interesting to note that in my case the first symptom pointing to accessory sinus trouble was the so-called congestion of the brain eight years ago, following pneumonia, and which was nothing more nor less

than an acute abscess of the frontal sinus. Nothing having been done for the relief of this condition, the bone eventually became extensively necrosed, the disease steadily progressing until the whole of the corresponding half of the ethmoid, both sphenoidal sinuses, and a considerable part of the sphenoid bone itself have been involved in the necrotic process.

The bacteriological examination of these cases is important, and should always be made with the view of determining the cause of this

FIG. 4.

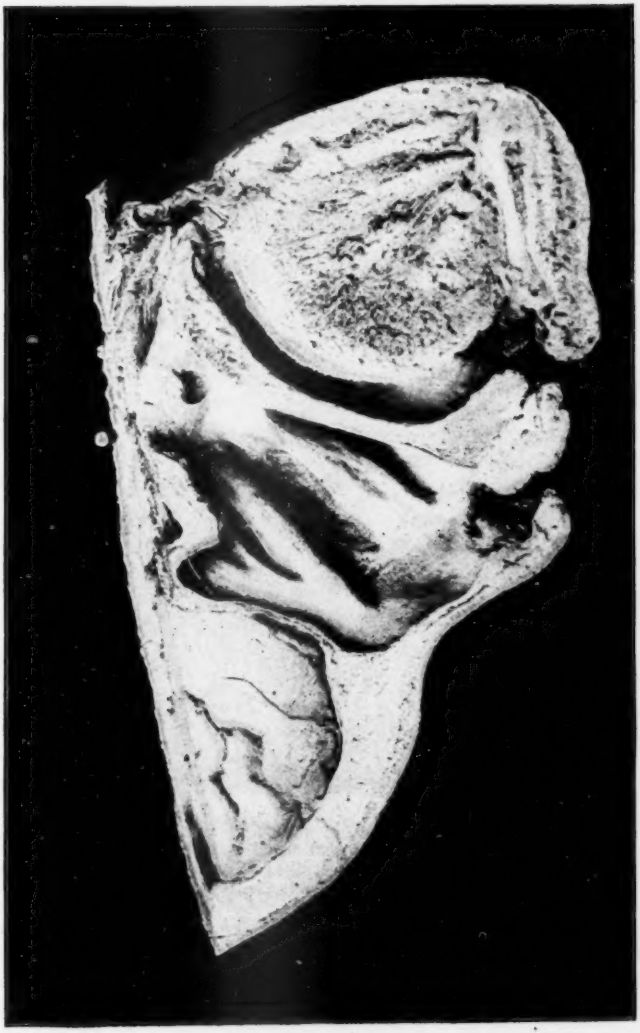
A photograph of a combined antero-posterior and transverse section, showing a very large left sphenoidal cavity with a very thick anterior wall bulging forward across the postnasal space.

little known affection. In two other cases, both of which terminated fatally, I neglected to have a bacteriological examination made. Both cases have been reported elsewhere, but a brief reference to them may be of interest in connection with this paper.

In one case both sphenoidal cavities were diseased in consequence of a neglected suppurative disease of the frontal and ethmoidal sinuses.

The patient died five days after an operation on the frontal sinus, an autopsy showing a meningitis resulting from an unrecognized abscess in the sphenoidal sinuses.

FIG. 5.

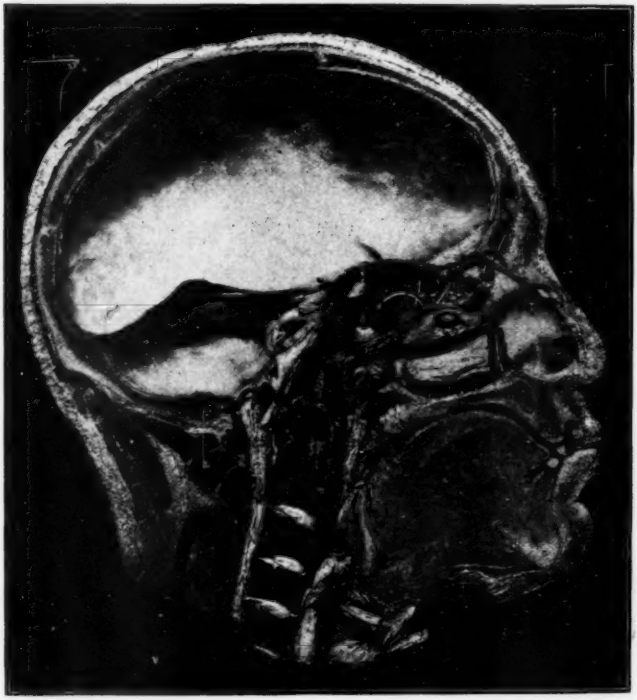


A photograph of the remaining portion of the section from the above preparation, showing a very small right sphenoidal sinus with its foramen presenting upward instead of forward.

In the second case a primary abscess of the sphenoidal sinuses was successfully opened, but during my absence from the city the patient died of meningitis, he having refused to remain in the hospital, where he could be under observation.

The sphenoidal cavities probably offer greater difficulties in the way of diagnosis and treatment than all the other sinuses, and the prognosis

FIG. 6.



A section with the middle turbinal partially removed, showing a very large anterior ethmoid cell, the middle ethmoid cells only partially developed, while the posterior ethmoid cell is quite a large cavity. The sphenoidal is unusually large in this specimen. F is a wire passing through the sphenoidal opening, the entrance into the posterior ethmoid cell, while B C D show three distinct openings into the frontal sinus. A is the entrance into the nasal duct.

is much more unfavorable, not only on account of the difficulty of keeping the cavities open, but also because malignant growths are much more frequently associated with them than with the other cavities of the nose. These cavities are subject to the same variations that we find in the other accessory sinuses. Although little has been done heretofore in bringing

out these irregularities, it is very important that they should be emphasized, in order that we may be on the lookout for them as a possible factor in cases that do not yield readily to treatment.

By far the best and safest method of opening the sphenoidal sinus is the one before mentioned; that is, after removal of the middle turbinal, with such a knife as used by Hajek or a modification of it, as shown in Fig. 1, a section is made in the anterior wall of the sinus, and with cutting forceps the opening can be enlarged sufficiently to meet the needs of the case. In a very narrow nose this method, however, might be impossible of execution; then that recommended by Jansen[2] at the International Medical Congress at Moscow might be tried. Jansen proposed to utilize the opening made in the maxillary sinus in cases of combined empyema of the accessory cavities, to open at the same time the posterior ethmoid cells, and next the anterior external wall of the sphenoidal sinus. The opening of the sphenoid is thus made easier and larger than by the nose, but it is often difficult and sometimes impossible, as remarked by the author, when the collection of blood is abundant, to distinguish between a large ethmoid cell and the sinus itself. This operation, I believe, has also been done successfully by Luc.[3] Furet[4] proposes the method of Jansen when the antrum is healthy and has practised it in one case successfully. He concludes that the nasal passages can be utilized in simple cases where the nose is sufficiently straight and large, and the maxillary route is indicated only in the following cases:

1. When the maxillary sinus participates in the inflammation.
2. In all inflammations of the sphenoidal sinus complicated with cerebral symptoms.
3. In all sphenoidal inflammations in subjects with narrow or abnormally formed nasal fossæ.

The new-growth in the case I have reported was first discovered about the latter part of September, 1900, and was probably the result of the irritation of the mucous membrane, whose vitality had been lowered by the prolonged inflammation of the adjoining cavities.

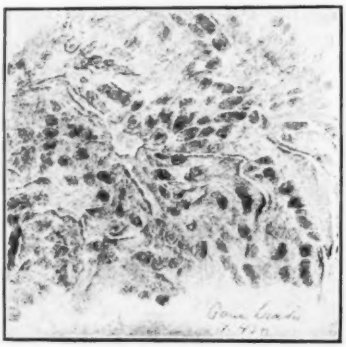
A part of the growth was sent to Dr. Jonathan Wright for diagnosis, and the following is his report: "Small, irregular masses of tissue; on microscopical examination prove to be made up of low-grade inflammatory tissue, the connective-tissue meshwork being filled not only with the products of inflammation, but with a typical proliferation of epithelium, often arranged in the forms of imperfect tubules and acini. In places there are irregular patches of epithelium and epithelioid cells, bloodvessels and true glands being almost absent. In all probability the growth had its origin in the glands of the mucosa. It is without doubt a malignant adenoma, and is apparently growing more rapidly than is the rule with these cases."

Pure adenoma is very rare in the nares; some authors, as, for instance, Bosworth,[5] deny that they ever occur within the nose. He states "that until further clinical evidence of the fact has been established we must conclude that unmixed adenoma does not occur within the nasal cavity." The mixed variety, however, as fibro-adenoma, myo-adenoma, angio-adenoma, papillary-adenoma, adeno-sarcoma, and adeno-carcinoma are relatively more frequent.

The principal features of these new-growths are their rose or grayish color, of firmer consistency than nasal polypi, rarity of epistaxis, and the preservation of a good general condition. These tumors are, according to Tissier,[6] generally benign, but may become malignant.

Upon reviewing the literature of this subject in the library of the Surgeon-General, U. S. A., I can find only five cases of unmixed

FIG. 7. FIG. 8.



Figs. 7 and 8 are drawings made from microscopical sections of the growth.

adenoma of the nasal fossæ reported, while of the mixed variety the literature is very much more abundant. Two of the five cases are referred to by Bosworth, viz.: those of Gosselin and Puglièse.

Gosselin's[7] case was a man, aged fifty-three years, who came under his observation in 1858; in the previous year, however, he had several polypi removed from the right nostril, by Fallin. When he came under Gosselin's care there was stenosis of the right nasal fossa, by a rather large, grayish, soft tumor, extending back into but not adherent to the nasopharynx. It was attached to the upper and anterior part of the fossa. The growth was removed by external operation, which was apparently successful. Microscopical examination showed the growth to be composed of glandular structures, with abundant epithelial cells. Gosselin refers to two previous similar cases, one at the Hôpital Cochin,

and one at the Hôtel Dieu. This later case was operated upon by Robin. After removal the patient finally died of meningitis, caused by the invasion of the ethmoid and sphenoid.

Puglièse's[8] case was a woman, aged sixty-five years; eighteen months previously she noticed a small induration corresponding to the lower part of the left lacrymal sac, simulating a commencing lacrymal tumor. It gradually enlarged, without material pain, causing exophthalmus divergent strabismus. There was no nasal stenosis, epistaxis, or discharge.

Microscopical examination showed it to be adenomatous, with some epitheliomatous degeneration.

The growth originated from the glands of the mucous membrane on the outer wall of the upper part of the nasal fossa; after enlarging it perforated the bone and penetrated into the orbit, the ethmoid cells, and maxillary sinus. It was removed by external operation, which was apparently successful, no recurrence having been reported.

MacKenzie Johnson[11] reports a case of true adenoma of the nasopharynx, in a boy, aged thirteen years; there was a fine sessile tumor in the nasopharynx, pale red, the size of a small walnut; it did not bleed, and gave the impression of adenoids. The growth was removed with forceps and the patient was well within one year. Microscopical examination showed the tumor to be a true adenoma, covered by a cylindrical epithelium.

Thomas J. Harris[9] also reports a case of true adenoma of the nose in a man, aged seventy-eight years. Patient had been under his care for two years, for nasal growths, which completely filled both nostrils and sprang from the ethmoid. The growths were removed with the snare, and the bases curetted. There was a partial recurrence within a month; then freedom for a year. In November, 1894, there was a further recurrence in the left nostril, with pain and exophthalmus on that side, and the patient died. Microscopical examination showed the growth to be almost pure adenoma, with scattered areas of small, round, and spindle cells.

H. Eichler[10] related the history of a case of adenoma simulating polyp of the nasal septum. The patient, whose age and sex are not given, was sent to him for nasal obstruction. Examination showed hypertrophy of the mucosa of the lower turbinates and a polyp of 1¾ by ¾ by ½ centimetre, attached to the septum on the left side, that was striking on account of its rosy-red color. It was removed with a snare. Microscopical examination showed it to be a pure adenoma.

Wright[12] says "that pure adenoma of the nose is almost unknown, although hypertrophy of the glandular structures of the mucosa is occasionally seen carried to such an extent that the question of its inflammatory origin may well be raised."

May 15, 1902. Since reading the report of this case at New Haven, a very decided improvement has taken place in the patient's condition. I have succeeded in removing the entire growth, and after an interval of one year there is at present no evidence of its return. The suppurative inflammation in the sphenoidal sinuses has ceased, and there remains only one small cell in the posterior ethmoidal region where pus is formed. Her general condition has improved to such a degree that she is able to attend to her household duties, and she is now leading a comfortable existence.

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TRAUMATIC RUPTURE OF CHORIOID, WITH HEMORRHAGE FROM UPPER BRANCH OF THE INFERIOR TEMPORAL VEIN.[1]

BY CHARLES A. OLIVER, A.M., M.D.,
OF PHILADELPHIA.

THROUGH the kindness of Dr. George C. Harlan, one of my colleagues at Wills Eye Hospital, I have been able to make a most careful ophthalmoscopic study of this most interesting case:

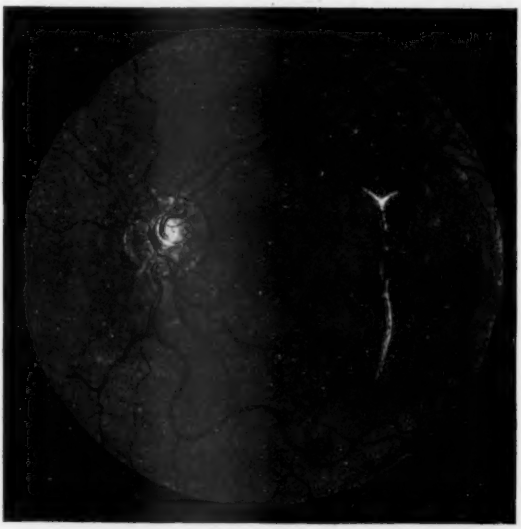
The patient, aged nineteen years, a waiter, was sent to the hospital by Dr. Harlan on December 23, 1898. The man gave a history of dimness of the vision of his left eye, dating back for one week, from having struck the eyeball against a protruding angle of a sideboard.

When I first saw him, an elongated Y-like break, situated immediately to the temporal side of the macula lutea, extending through the chorioidal membrane directly down to the scleral coat, was plainly visible. Running almost vertically upward in the split area and coursing over the corresponding artery, the upper branch of the inferior temporal vein could be seen. At a position in this break corresponding with a point just beyond the macular region there was a fresh hemorrhagic extravasation from the vein, which was commencing to undergo absorption. The most pronounced absorbing areas in the extravasation,

[1] Read before the Section on Ophthalmology of the College of Physicians of Philadelphia.

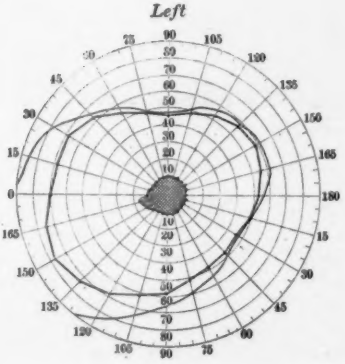
which were the thinnest and the least dense in the blood mass, consisted in four elongated finger-like tips that were situated on the inner surface

FIG. 1.



of the retinal sheet. The borders of the break were sharply outlined and were devoid of any pigmentation. The rest of the eye-ground, with

FIG. 2.



the exception of an almost imperceptible grayish haze just above and including the macular region, appeared normal.

These appearances are very well represented in Fig. 1, which

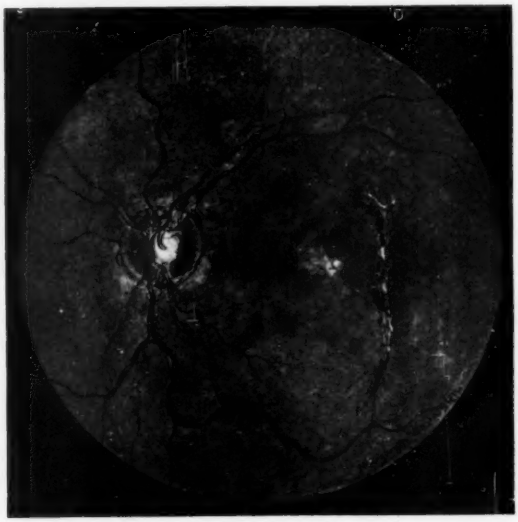
has been reproduced from a water-color sketch made at the time by Miss Margaretta Washington, of this city.

In all other respects the eyeball was apparently uninjured and intact. Central vision was markedly reduced (one-fortieth of normal), this being accounted for by the presence of an absolute central scotoma of about eighteen degrees in its long and ten degrees in its short diameter. (Fig. 2.) The extent of the visual field for both form and color was but slightly and concentrically reduced from the normal average. Both extra-ocular and intra-ocular muscle-actions were undisturbed.

The fellow-eye was healthy and performed its various functions properly.

One week later the fundus changes in the affected eye had become so different that a second sketch (shown in Fig. 3) was made by Miss

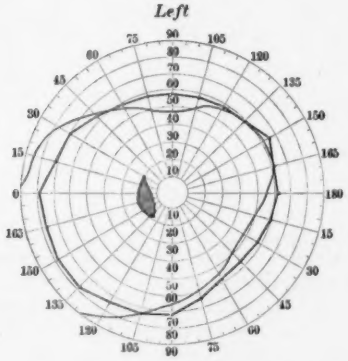
FIG. 3.



Washington. As may be readily noticed, the ophthalmoscopic appearance of gross inflammatory reaction in both the chorioidal and the retinal coats was brought into view. The fundus-reflex, particularly in the papillo-macular region, had undergone a decided change, and the surface of the retina had become very irregular. Areas of retinal thickening and opacification; elevated points of deeply seated hemorrhages; chorioidal thinnings and commencing degeneration; pigment aggregation around the borders of the most disturbed portions of the two coats, with marked retraction of the edges of the slit-like rupture, which had become serrated, constituted the main details of a picture which was in gross contrast with that of the previous week. If to these changes an engorgement of the main venous trunks, a broad yellowish halo of retinal opacity around the optic nerve-head, and a pronounced degree of lessening of the large retinal hemorrhage in the

linear rupture, be added, a series of most characteristic peculiarities that may be considered as expressive of the gross inflammatory stage of the condition are obtained.

FIG. 4.



In spite of these changes the field of vision for white (as shown in Fig. 4) had become somewhat larger, and the scotomatous area had

FIG. 5.

become much smaller and had removed outwardly so as to entirely include the physiological blind-spot. Vision had risen to two-fifths of normal.

On January 16, 1899—seventeen days after the date of the making of the second water-color drawing—a third sketch (as shown in Fig. 5) was made by Miss Washington. In it it will be found that the hemorrhage in the rupture had disappeared, the previously underlying retinal vessel wall could be plainly seen as intact, and the contained venous portion of the current was uninterrupted in its flow. The large area of localized œdema, which was situated just above the macula and which had become partially opaque, was replaced by a semi-atrophic spot. Midway between this area of degeneration there was a linear atrophy which had been represented in the second sketch by a short vertical line of deeply-seated pigment aggregation. The entire papillo-macular region, particularly along the borders of the areas of atrophy, was much more densely pigmented than before, and had become yellowish in many places. The yellow opaque halo around the optic disk had broadened and apparently had become more consistent. The fine macular twigs of the retinal artery series still pursued their undisturbed courses over the large atrophic area, while the previous engorgement of the main vascular stems of the retinal circulation had subsided, and the tint of the contained venous blood had again become normal.

The visual field exhibited a persistency of the excentrically placed positive scotoma, and central vision remained practically the same as before.

Remarks. The case has been considered worthy of record upon account of the great rarity of opportunity to obtain graphic studies of three of the most important stages in the disease, stages that are so unlike and so rapid in their onset and so brief in their duration.

REPORT OF THREE CASES OF CREEPING LARVÆ IN THE HUMAN SKIN (HYPONOMODERMA, KAPOSI).

By Arthur Van Harlingen, M.D.,
of Philadelphia.

The number and variety of animal parasites of the human skin are probably greater than has been generally supposed. On account of the rarity of such cases, however, they frequently go unrecognized or are mistaken for other affections of the skin.

The cases I am about to describe were met only at long intervals, and in an extended experience I have not encountered any others. Two of them occurred in children. The third case was that of an adult, and has been published before under another name, but I shall cite it as illustrating an affection which in this country and climate must be very rare.

Case I.—Harriet V., aged four years, was brought to the Howard Hospital, on October 21, 1884, with an eruption on the foot of three or four weeks' duration. According to the account given, this eruption

had originated on the heel in the form of a small water-blister, which had gradually extended on one side while healing on the other, following a serpiginous course up and inside of the foot just in front of the heel, and terminating in a spiral in the skin over the inner malleolus.

The appearance of the lesion was that of a line of small vesicles resembling, in a magnified degree, the furrow made by the itch insect. The lesion was about four inches in length and about one-sixteenth of an inch wide. It was extremely itchy.

The patient was directed to have the skin well rubbed with an ointment of sapo viridis and tar along the line of disease. Under this treatment the lesion gradually disappeared, until, at the end of four weeks, not a trace remained.

FIG. 1.

CASE I.

I felt certain that the disease in this case was caused by an animal parasite. The peculiar beaded appearance, due to the string of small vesicles containing black granules—probably débris or fecal matter—was, as has been said, very much like the furrow of the itch insect, but on a larger scale. The itching sensation was also very characteristic. This is commonly most marked during the activity of the parasite where the furrow is extending rapidly, but the fact was not particularly noted in this case.

CASE II.—John W. K., aged five years, was brought to the Children's Hospital, on October 23, 1901, displaying an eruption on the sole of the foot, consisting of a flat, red, narrow line curving from one side of the foot to the other, and resembling the edge of a circinate erythema, for which, indeed, I mistook it at first sight. The child's mother, however, persisted in the assertion that there was a worm beneath the skin, which at times was prominent and at other times seemed to sink out of sight, changing its locality also from time to time, and meandering over the foot from one place to another.

Unfortunately, the mother's statement was weakened by a description which she gave of the worm, saying that it "seemed about an inch long, having eyes and a prominent head." This statement was

evidently imaginative. There had been some inflammatory symptoms, and there was considerable itching in the part, especially at night.

No history pointing to exposure could be obtained. The child did not go barefoot while in the city, but during the previous summer, while at Atlantic City, he had run about upon the sand without shoes or stockings, and had suffered, his mother said, from a slight cut in the sole of the foot at the point where the disease was afterward first noticed.

FIG. 2.

CASE II.

The patient was kept under observation, but for some time no change appeared in the red line, which extended across the sole of the foot. When seen on December 26, however, the appearance of the lesion had decidedly changed. It now appeared raised and of a brighter red, like a whip-cord under the skin, and the child complained of pain in the part.

Within the next few weeks the lesion had made considerable progress, extending at one end, running around the side of the foot, and pushing its way in an irregular line over the dorsum and toward the bottom of the little toe.

The progress of the parasite was so rapid at this time that a few hours would suffice to show perceptible progress. Dr. Siter, at my request, cut down on the extremity of the red furrow, hoping to intercept and extract the parasite, but no living organism could be found.

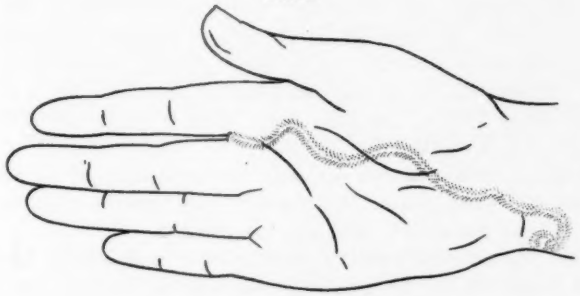
The patient disappeared from view after this, but on April 4, 1902, I called upon the mother, and found that immediately after the little operation the red line began to fade away, and in a short time had disappeared. Up to the present time, although three months have elapsed, there is no tendency to a recurrence of the disease.

It is possible that Dr. Siter's incision may have exposed the parasite, which was washed away in the bleeding that followed.

The third case I have to report occurred in an adult, but as it illustrates the affection under consideration I will give an abstract of it.[1]

CASE III.—H. C. B., an employé in the Custom House, came to my office on January 17, 1888. He said that he came in contact with

FIG. 3.



CASE III.

invoices from all parts of the world, but did not handle foreign goods. He stated that he never lived outside of Philadelphia. The affection had first showed itself three months previously in the form of a small, red, itchy pimple on the back of the hand. This soon began to extend and elongate, taking on a serpentine form and pushing along under the skin, which it elevated in its passage, leaving a red, cord-like elevation of the epidermis. The late Prof. Agnew, under whose care the patient was at that time, used hypodermic injections of a 50 per cent. solution of carbolic acid, but without effect, and the parasite continued its course, running between the fingers, then diagonally across the palm of the hand, and finally terminating in a whorl-like curve over the wrist-joint, to which point it had reached when I first saw it. Under the thinner skin the lesion looked like a vesicular eruption.

I must confess that in this case, also, I was at a loss to make a diagnosis, and finally decided upon one which I now believe to have been erroneous, namely, that of dracunculus or filaria medinensis. Under

[1] The case was published in the Medical and Surgical Reporter, October 6, 1888, under the name of "filaria medinensis," a misnomer.

this belief I cut down upon the supposed worm at various points in its course, but, of course, without finding it. I then prescribed asafœtida, which has been used successfully in filaria medinensis, under which the red cord rapidly sunk to the surface, turned brown, and faded away.

As there was still some slight trace of irritation at the extremity of the lesion when I last saw the patient, I am not perfectly sure that a cure was effected. The patient, however, was of the opinion that the parasite was dead.

The cases just described are, I believe, the first to be reported in this country. A few similar ones are to be found in European medical literature.

So long ago as 1870 Lee[1] reported under the title of "Creeping Eruption" the case of a child, aged three years, who had first shown signs of the disease three weeks previously. A red line was noticed by the mother just below the right ankle. This gradually travelled up the leg and thigh and onto the abdomen, meanwhile fading on the leg. There was no pain or itching. On examination a pale rose-pink line, one-eighth to one-sixth of an inch in breadth, could be seen coursing across the abdomen below the umbilicus, presenting several loops. In some places this line had a stippled character, like a number of dots, rather than a continuous line. (This is similar to the appearance shown in my Case I.)

A piece of skin was cut out near the termination of the line, for microscopical examination. No parasite was found, but the red line began to fade immediately, and soon disappeared.

Later, Lee[2] reported a similar case occurring in an adult. Here the disease began in the buttock, and was supposed to have been contracted in a water-closet.

Walter G. Smith[3] described a somewhat similar case beginning as a swelling in the outer ankle of the right foot, painful, and moving slowly up the leg and body to the right axilla, right breast, and back of the neck. The line then terminated in an abscess, which opened and gave exit to a dipterous larva, 9 mm. in length and 2 mm. in diameter, of which Smith gives a figure. Several similar larvæ were afterward observed in this same case, each pursuing a course similar to the first one.

I am inclined to think this affection due to some different parasite from that causing the eruption in my cases. The ending was different, as none of my cases showed a terminal abscess; nor do I think it possible that such a large parasite could have been the cause of the narrow line; it would have excited more irritation than was observed in my cases.

[1] Transactions of the Clinical Society, vol. viii. p. 44.
[2] Ibid., vol. xvii. p. 75.
[3] Transactions of International Medical Congress, 1881, vol. iii. p. 181.

Under the title of "Larva Migrans," Crocker[1] reports a case where as much as seven inches' progress was made in a day. Repeated efforts were made to capture the supposed larva by cutting out pieces of skin at and beyond the end of the red line, but without avail.

Kaposi[2] gives illustrations of the affection, and calls it *hyponomoderma*, from ὑπόνομος and δερμα, "that which burrows under the skin"—a non-committal term. Another name given to the disease is *myiasis linearis*. He has only seen four or five cases in his enormous experience, but the affection is said to be more common in certain parts of Russia and in Arabia.

It is unfortunate for the completeness of these cases of mine that I have been unable to demonstrate the larva supposed to have caused the skin symptoms. No better fortune, however, has attended the efforts of other observers, and the particular parasite which gives rise to the skin phenomena has never been demonstrated.

It will be noticed that erroneous diagnoses were made in two of my own cases at first. The affection is at times quite difficult to make out, as its appearance changes considerably. Lee's case was examined by several experienced dermatologists, including Sir Dyce Duckworth, but their opinions were wide of the mark. The discovery of the particular larva producing the affection is a great desideratum.

BENIGN CYSTIC EPITHELIOMA: REPORT OF TWO CASES PRESENTING UNUSUAL FEATURES.

By M. B. Hartzell, M.D.,
INSTRUCTOR IN DERMATOLOGY, UNIVERSITY OF PENNSYLVANIA; DERMATOLOGIST TO THE PHILADELPHIA HOSPITAL.

The two following cases of benign cystic epithelioma which I have had the opportunity to study with some detail within the past two years present clinical types of this interesting affection which, so far as I have been able to learn from a careful review of the literature of the subject, have not yet been described.

Case I.—Mrs. E. K., a woman, aged about eighty years, whom I saw through the courtesy of Dr. W. D. Robinson, her family physician, had a rounded, dollar-sized patch of nodules, made up of several smaller, coalescing groups arranged in an irregularly circinate manner around scar-like areas, situated on the right side of the forehead, over the frontal eminence. These nodules, for the most part confluent, were the size of a hempseed, firm, translucent, bluish-white, with a small blue-black dot in the centre of each, resembling a minute gunpowder

[1] Diseases of the Skin, Philadelphia, 1893, 2d ed., p. 926.
[2] Harnkrankheiten, Aufl. 5, 1899, p. 1039.

stain. Near the centre of the patch was an oblong, superficial ulcer covered with a thin crust. The patient stated that the disease had lasted six or seven years, beginning with a single lesion "like a mosquito-bite," and spreading slowly by the addition of new nodules rather than the enlargement of the old ones, which, after reaching a certain size, remained stationary. The central ulcer was of much more recent date, having appeared about two years ago. There was no pain or any other marked subjective symptom. No similar lesions were present on any other part of the body.

FIG. 1.

For the opportunity to study the second case clinically and microscopically, and for the use of his notes, I am indebted to Dr. Charles N. Davis, under whose care the patient had been for some time.

CASE II.—F. G., a nurse by occupation, aged thirty-eight years, in excellent general health, had six or seven dime- to half-dollar-sized oval and irregularly shaped, sharply limited patches, consisting of central areas of scar-tissue and atrophied skin surrounded by a bead-like border of pinkish and yellowish large pin-head to split-pea-sized nodules, situated on the upper part of the back and chest. Three of these patches were over the right scapula, one over the left, and two beneath the left clavicle. The nodules were quite firm and slightly translucent, as if containing fluid, but none was obtained upon punc-

turing them. Scarring was present only in the larger patches upon the chest, where an unsuccessful attempt had been made to destroy them with the curette; but the atrophy in the remaining patches was a part of the disease process, the patient stating most positively that no treatment of any kind had been applied to the lesions upon the back. It should also be noted that in the curetted patches new nodules had made their appearance at the border of the cicatrix. The affection was not attended by subjective symptoms of any kind, and had lasted four or five years.

Lesions were excised in both cases for microscopical examination—in the first case from the border of the patch upon the forehead, and in the second from both the chest and back. The microscopical

FIG. 2.

appearances corresponded very accurately with those described by previous observers, and were practically the same in both cases. The epidermis presented no abnormality except a slight thinning in places where it had been subjected to pressure from below by the cell-masses about to be described. In the upper and middle portions of the corium were numerous round, oval, and irregularly shaped masses of columnar and oval epithelial cells, the greater number of which contained a central cavity in which was a quantity of granular débris evidently resulting from degeneration of the centrally situated epithelium. (Fig. 2.) About the borders of many of these cell-masses the cells were of the columnar variety, and were arranged radially, presenting a palisade-like appearance. They stained much more deeply

than the oval cells in the centre of the mass, many of which took the stain feebly or not at all. A considerable number of the round and oval cell areas, when followed through a series of sections, were found to be connected with the basal columnar cell layer of the epidermis, and had evidently taken their origin from the cells of this layer. Besides the round masses there were others presenting a great variety of irregular shapes, sending out branching processes and containing curiously tortuous cavities in their interior. Followed through a series of sections they could, in some instances, be traced to hair follicles, from which they seemed to have originated. In some of the sections made from a nodule taken from a patch upon the back of the

FIG. 3.

second case a large epithelial tract presenting a reticulated arrangement was seen, resembling to some degree the structure of an angioma, but the connection of this mass with the epidermis was easily demonstrated. No such arrangement of the epithelium was observed in any of the other lesions examined. In the small epithelial masses the central cavity was correspondingly small, and was filled with an amorphous material exhibiting the staining reactions of colloid matter. In the largest cysts observed in sections of the lesion taken from the first case a considerable quantity of brown, granular pigment was noticed, but none was observed in the sections of the second case. (Fig. 3.) Around the epithelial cell-masses there was a moderate round-celled

exudate, and in some instances a well-marked ring of fibrous tissue separated them from the fibrous tissue of the corium. The sebaceous and sweat-glands were in appearance altogether normal.

These two cases present certain noteworthy peculiarities. In all those hitherto reported the nodular lesions were scattered about over the face and upper part of the trunk without any special arrangement; nothing like the circumscribed patches described above was observed. The advanced age, too, at which the disease first showed itself in one of the cases was decidedly unusual, as in all those previously described it first appeared early in life, commonly within the first two decades. The ulceration which occurred in this case is likewise worthy of special note, although this has been previously observed by Jarisch, White, and Hallopeau, each reporting a case in which ulceration resembling that of ordinary epithelioma took place, but the scar-like atrophy common to both cases has not hitherto been observed.

In 1892, under the name epithelioma adenoides cysticum, Brooke reported four cases of a cutaneous disease, all occurring in the same family—a mother and three daughters—characterized by a nodular eruption confined almost exclusively to the face, there being only a very few scattered lesions upon the shoulders and upper part of the arms. Most of the nodules contained one or more milium-like bodies, and the eruption was so abundant in places as to form lumpy patches on various parts of the face. Microscopical examination of the lesions showed that they were composed of an epithelial new-growth in the corium, which in some sections resembled a sweat-gland adenoma. There were numerous epithelial tracts coiled on themselves, forming masses of various shapes in which were circular or oval cavities filled with colloid matter or horny epithelium. In other sections dense cell-masses predominated, there being few tube-like tracts. In all cases the cell-masses were separated from the surrounding connective tissue by one or more layers of palisade-like cells, which stained more deeply than the cells in the interior of the mass. The connective tissue about the epithelial masses was finer than normal, forming a dense capsule. In some of the sections the neoplastic epithelium was connected with the epidermis and with the hair sacs, and the author believed these structures to have been the piont of origin of the tumors. These cases were regarded by Brooke as being identical with the cases of Jacquet and Darrier and others, which had been previously reported under various names, to which reference will again be made.

About the same time Fordyce reported two cases, occurring in a mother and daughter, resembling in all their clinical and microscopical features those described by Brooke. There was the same nodular eruption confined to the face and neck, and the same epithelial new-growth in the corium. Some of the tumors were made up almost

entirely of densely packed epithelial cells, while in others the epithelium was arranged in bands and tracts intricately intertwined. In still others there were "pearls" and "cell-nests," as in ordinary epithelioma. In some instances the cell tracts presented an alveolar structure resembling glandular tissue. A direct connection of the cells of the new-growth with the columnar cells of the epidermis and with the external root-sheath of the hair was observed in some of the tumors, and the author thinks it evident that the epithelial masses forming the tumors were the result of the proliferations of these structures.

In 1894 Jarisch described a case of eight years' duration, occurring in a young man, aged twenty-two years, in which, besides a nodular eruption situated upon the eyelids, glabella, and temples, there were several small ulcers resembling ulcus rodens. No similar lesions were present upon any other part of the body. Microscopical examination of the nodules and ulcers revealed an epithelial deposit in the *pars papillaris* and the upper part of the *pars reticularis*. These occurred partly as round-cell masses, partly as branching cell tracts tending toward a central point which either enclosed a hair or concentrically layered, flattened, cornified cells. These cell-masses and tracts were separated from the adjoining connective tissue by one or more layers of darkly stained, cylindrical cells. The cells within this palisade-like border varied from round or polygonal to spindle shape, the former being found in the broad masses, the latter in the slender tracts. The connection of the cell tracts with the hair follicles could be established with ease, but no such connection with the epidermis or sweat-glands was observed. In some of the sections the cell-masses seemed to spring directly from the epidermis, but when these were followed through a series they were always found to be connected with hair follicles. Although the case was regarded by Jarisch as being identical with those described by Brooke, he gave it a new name—tricho-epithelioma, as indicating its origin from the hair follicles.

White has reported the case of a woman, aged forty-five years, with some fifty papules and tubercles upon the face, varying in size from a pin-head to a dime and even larger, the small lesions being the color of the skin, while the larger ones were much redder. A number of the medium-sized tubercles were either depressed in the centre or had begun to soften, and were covered with brown crusts. Two or three lesions were much more prominent than the rest, with abrupt, perpendicular edges and depressed centres, like Hutchinson's crateriform epithelioma. On the right upper eyelid, on the inner portion of both lids of the left eye, and on the side of the nose were open ulcers presenting the characters of rodent ulcer. Besides the lesions on the face there were a few on the neck, shoulders, and forearms, but none of these had undergone any secondary change. Bowen, who examined

the tubercles microscopically, found epithelial masses in the corium arranged in islands and elongated tracts, the latter being connected and intermingled in a complicated way. In the midst of the epithelial islands and bands were small cysts with granular or homogeneous contents; in a few, corneous material, either alone or with colloid, was present. In all the tumors connection of the epithelial mass with the lower cells of the rête could be demonstrated. No proliferation of any of the glands of the skin was found. This case is remarkable for the secondary ulceration and the unusual size of the lesions.

Hallopeau has likewise reported a case, although under another name, in which ulceration resembling that occurring in ordinary epithelioma took place. The diagnosis apparently was entirely clinical, as no account of the histology of the lesions is given. The eruption was situated upon the lids, and had existed since infancy.

Wolters has recently reported a case which differs clinically from those previously reported in that there was but a single lentil-sized, yellowish-red tumor over the right eyebrow. The patient was a girl, aged twenty years, and the lesion had existed since birth. It was composed of a maze of cell tracts containing large and small cysts. The epidermis was everywhere intact, but the interpapillary pegs were unequally developed and of varying shapes, and many of them were in direct connection with the cell tracts running through the cutis in all directions. The cysts contained either homogeneous colloid masses or horny, laminated globes. Besides slender epithelial tracts there were others, thicker and more massive, which seemed here and there to be connected with hair follicles, and which, like the others, contained cysts. Most of the epithelial tracts originated from the interpapillary pegs, but their origin from the hair-sheaths could also be demonstrated. The sebaceous and sweat-glands and the bloodvessels were normal.

Within a few months Pick has described a case which seems to be intermediate between benign cystic epithelioma and adenoma sebaceum, presenting some of the features of both neoplasms. A man, aged forty-three years, with a marked acne rosacea, had an eruption of small nodules seated upon the temples and forehead. There was likewise a slightly depressed, atrophied patch over the left zygoma. The nodules were found upon section to be composed of variously sized and shaped epithelial cell-masses partly anastomosing with one another. These epithelial new-formations began immediately beneath the epidermis, and extended, with their processes, down to the fat tissue. Their lobed structure recalled that of the sebaceous glands. The external border of the new-formed cell-masses was formed by cells arranged in a palisade-like manner, with well-stained nuclei. These cells were in all respects like those of the basal layer of the epidermis, with which, when the cell-masses joined the epidermis, they insensibly

blended. In the cell-masses were variously sized cysts, occasionally empty, sometimes containing a mass staining intensely with acid dyes (eosin, picric acid) or finely granular material. The sweat-glands showed no abnormality. The histological picture corresponded with that presented by the cases of Brooke and Fordyce. The author concludes that the case was one of multiple adenoma of the sebaceous gland, corresponding to the sebaceous gland nævi, and that, upon the basis of such an adenoma, epithelial tumors showing the histological peculiarities of epithelioma adenoides cysticum were formed by the proliferation of the border epithelium of the sebaceous gland acini.

In connection with this case reference should be made to the two cases of nodular eruption of the face and scalp reported by Balzer and Ménétrier, and Balzer and Grandhomme as adenoma sebaceum, since they were most probably examples of benign cystic epithelioma. Microscopically, the tumors were found to be composed of proliferating epithelial tissue arranged in lobules containing cysts, the seat of the neoplasm being the pilosebaceous apparatus.

In all the cases thus far referred to the lesions were practically confined to the face, and in all the microscope showed an epithelial new-growth having its origin in one or more of the epithelial structures of the skin.

In 1887 Jacquet and Darier described for the first time a disease of the skin characterized by an eruption of papules and tubercles confined exclusively to the anterior surface of the trunk and inner surface of the upper extremities. Darier, who examined the lesions, found that they were composed chiefly of a great number of epithelial tracts situated in the middle portion of the derma, cylindrical in shape, and ramifying in all directions. Almost all of these epithelial tracts presented globular, cystic dilatations containing blocks of an amorphous, refracting substance. The arrangement of the epithelial tissue, its localization, and the multiplicity of the tumors led the authors to regard the affection as an adenoid epithelioma; and, although not able to certainly trace a connection with the sweat-glands, they concluded that the neoplasm originated in these structures. As indicating the origin of the lesions they designated the malady eruptive hydradenoma. Two years later, at the Paris meeting of the International Congress of Dermatology and Syphilis, Jacquet exhibited a second case presenting the same clinical features, but he denied the relationship of the disease to the sudoriparous apparatus, believing that the nodules were epithelial tumors developed from "erratic para-epithelial débris," and proposed a new name for it—benign cystic epithelioma. Upon the same occasion Quinquaud likewise presented a case in which there were hundreds of nodules, limited, as in the other cases, to the trunk. He expressed himself as being uncertain as to the origin of the neoplasm,

but thought that it might have started from aberrant epithelial cells. At a later period Philippson reported two cases, and Torok one from Unna's clinic. The former regarded the tumors as benign epitheliomata with colloid degeneration, and endeavored to identify the affection with colloid milium; the latter considered the disease cystadenoma which had originated in the sweat-glands. Jarisch and, quite recently, Wolters have reported cases which they considered identical with those of Jacquet and Darier, Quinquaud, Philippson, and Torok, clinically and microscopically; but they denied the epithelial character of the malady, being able, as they believed, to trace the new-formed cell-masses to the endothelium of the bloodvessels. These authors regard the affection as quite distinct from benign cystic epithelioma.

From the foregoing brief summary it is apparent that more or less confusion exists as to the exact relationship of the cases of this last group to benign cystic epithelioma and to one another.

In conclusion, it seems probable that all these cases, described under such a variety of names, represent three affections, presenting lesions which can only be distinguished from one another with the aid of the microscope, but which are quite distinct in their nature and origin: First, benign cystic epithelioma, in which the lesions are usually, but not always, situated upon the face, and have their origin in the basal layer of the epidermis and the external root-sheath of the hair; second, cystadenoma, in which the eruption occurs upon the trunk, the tumors starting in the sweat-gland apparatus; and third, hæmangio-endothelioma, likewise situated upon the trunk, but originating in a proliferation of the endothelium of the bloodvessels.

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POST-OPERATIVE NON-SEPTIC LEUCOCYTOSIS AND OTHER BLOOD CONDITIONS.[1]

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The following observations were undertaken, in the first instance, for the purpose of establishing a normal standard of leucocytosis in non-septic post-operative wound repair. It was thought that could such a standard be established, any material departure from it, following an operation, might be regarded as an early sign of post-operative sepsis, and possess a clinical value accordingly.

The first series of observations in ten cases was begun in the autumn of 1898, and completed prior to the annual meeting of the Michigan State Medical Society in May, 1899, at which meeting, by request, I presented a preliminary report, which was published in the *Transactions* for that year. In this first series the chief interest attaches to the rise and fall of the post-operative leucocytosis, a differential count of varieties of leucocytes not being attempted. It was observed, however, that the numerical value of the red blood corpuscles as well as the hæmoglobin *rose after operation*, even where considerable blood had been lost at the time of operation, and this led, in the second series, to a more systematic study of the red elements and hæmoglobin, for the purpose of ascertaining whether this increase in peripheral blood value following operation was real or apparent. At the same time the leucocytes were studied not only as a whole, but in the relation of their varieties to each other. If by blood examination it were possible to differentiate between an aseptic wound repair and the commencement of a septic process following operation at a time sufficiently early to be of practical value to the surgeon, a study of the blood in all surgical cases of major importance would be the rule of practice; and while the conclusions to be gathered from the records of my two series of cases are perhaps not sufficient for the establishment of fixed standards, the findings in certain respects appear so constant as to warrant their acceptance as *rules* in the pathology of post-operative wound-repair.

I am by no means so certain of the *clinical* importance of blood examinations in these cases. It was thought that such examinations systematically made would show a marked "jump" from the leucocytosis (if it existed at all) of the aseptic process to the familiar blood picture of sepsis, and that at a time so early as to be of practical value. My observations seem to have demonstrated two points of some importance in this regard:

[1] Read before the Chicago Pathological Society, May 12, 1902.

First, that the leucocytosis of a normal wound-repair (even where healing is rapid and by first intention, when no drainage is employed, as in simple laparotomy for tuberculous peritonitis) may be very marked; and second, that the "jump" in degree of leucocytosis between the two conditions does not exist at all.

On the other hand, these experiments demonstrate that at a time subsequent to operation, when the results of infection are most prone to appear, the leucocytosis of normal wound-repair is on the decline; and that if, therefore, the leucocytes appear in increasing number at such a time it may safely be assumed that infection has occurred, such other morbid conditions as effect similar changes in the constituent elements of the blood being eliminated. Further, that while in the blood of the healthy individual there is a standard average number of leucocytes (7500 to 1 c.mm., the different varieties occurring in practically uniform proportion), it is found that the subject for the operating-table, while not necessarily septic, is, of course, not in a normal condition of health, and will therefore present, very frequently, marked variations from this average, associated or not with anæmia or chlorosis. A count made prior to operation will, therefore, represent the "special normal" for each particular case to be observed, which it is of the first importance to ascertain if any significance is to be attached to the post-operative observations. The relative proportion between the various forms of leucocytes is also found to be very inconstant in such conditions as usually demand surgical relief, but this fact is of less importance to the present purpose.

In its relation to septic infection, then, a single examination of the blood made subsequent to operation is of little value either to pathology or diagnosis, since it may present a leucocytosis perfectly normal to one patient which would be quite as abnormal to another. A series of observations commencing six hours or more prior to operation and carried through the period when sepsis may be a possible element of danger will, on the other hand, convey much that is significant to the progress of the wound-repair.

The method of observation adopted in my first series of ten cases was as follows:

First. A complete quantitative examination of the blood (exclusive of a differential leucocyte count) was made from six to twenty hours prior to operation. (In Case VI., first series, this examination was made three days prior to operation, the delay being occasioned by the appearance of menstruation, and a second leucocyte count was made seventeen hours before operation.)

Second. A leucocyte count was made six hours after completion of operation.

Third. A second quantitative examination similar to the first was

made twenty-four to thirty hours after operation except in the first two cases.

Fourth. A leucocyte count was made every twenty-four hours thereafter until all danger of sepsis had passed and the leucocytes had fallen to practically a normal number.

The same instruments were employed in all cases (the Thoma-Zeiss hæmocytometer and von Fleischl's hæmoglobinometer). My pipette for red corpuscles counts rather lower than it should; but as the same pipette was used constantly, and comparative observations are all that are necessary, the results meet the requirements.

In estimating "color index," 5,000,000 per cubic millimetre of red corpuscles was taken as the normal 100 per cent. This is rather higher than normal for women, but a single standard for all cases was considered advisable. Like conditions of nutrition were observed in all examinations, and by reason of the surgical nature of the cases, of course, digestion leucocytosis was always to be excluded from consideration.

The greatest possible care was exercised in procuring the specimens, diluting and mixing the same, and counting; but as absolute accuracy is not obtainable with the Thoma-Zeiss apparatus, note was not made of anything under 100,000 per cubic millimetre in counting the red cells, nor under 100 in the colorless elements.

The blood in all cases is peripheral and obtained from the finger-tip by a puncture sufficiently deep to allow of procuring the specimen without compression.

Such other precautions as were possible were taken to avoid errors in technique.

For obvious reasons, major operations alone were selected for the purpose, both in the first and second series. In the first series of ten cases all were abdominal sections except one, which was an amputation of the breast for malignant disease. One of the abdominal operations was also for malignant disease. None of the second series was a malignant case. Neither of the malignant cases presented evidence of cachexia, which, had it been present, would, of course, have rendered them valueless for the present purposes on account of the leucocytosis which characterizes the blood in such cases.

Notwithstanding the nature of the disease in a few instances in both series, so far as could be determined the general health in all cases was fairly good, with one exception (Case VII., Series II.).

There had been no history of recent suppuration, nor was any pus present in any of them at the time of operation nor subsequently during the periods of my observations.

Of the first series, nine were operations upon adult women, and one (the appendectomy) upon a vigorous young man.

Of the second series of seven cases all were abdominal sections in adult women.

The several operations in the two series were as follows:

SERIES I. Case I., abdominal section for oöphorectomy.

Case II., abdominal section for oöphorectomy (malignant).

Case III., abdominal section for ventrofixation.

Case IV., abdominal section for ventrofixation.

Case V., abdominal section for oöphorectomy.

Case VI., abdominal section for oöphorectomy.

Case VII., abdominal section for appendectomy.

Case VIII., amputation of the breast and removal of the axillary glands (malignant).

Case IX., abdominal section for removal of papillomatous cyst.

Case X., abdominal section for ovarian cyst.

SERIES II. Case I., abdominal section for removal of the tubes and ovaries.

Case II., abdominal section for ventrofixation and breaking adhesions.

Case III., abdominal section for removal of the tubes and ovaries.

Case IV., abdominal section for oöphorectomy.

Case V., abdominal section for gallstones.

Case VI., abdominal section for ventral hernia.

Case VII., abdominal section for abdominal tuberculosis.

The method adopted in the second series of observations, as above stated, is somewhat more exhaustive than that in the first, and consists in the following routine:

First. A quantitative examination, including a differential computation of leucocytes and the estimation of the proportion of nucleated erythrocytes, is made a few hours prior to operation. This, of course, necessitates the hardening and staining of cover-glass preparations.

(This additional and rather monotonous labor was suggested by the red-cell finding in the first series, where the second general examination, made from twenty-four to thirty hours after operation, invariably showed increase, at times startling, in the number of erythrocytes per cubic millimetre, and usually also in the hæmoglobin. It was thought that if this increase were real—*i. e.*, due to stimulation of the hæmogenetic function—there would appear with the increase in erythrocytes a proportional increase in nucleated red cells.)

Second. A leucocyte count is made six to twelve hours after operation.

Third. A second general examination similar to the first is made from twenty-four to thirty hours after operation.

Fourth. A leucocyte count is made every twenty-four hours following, as in the first series.

Fifth. A final general examination similar to the first is again made one hundred and twenty hours or more after operation, chiefly for the purpose of determining whether the apparent increase in erythrocytes is sustained.

To better illustrate the changes occurring in the blood in the course of these examinations four charts have been prepared.

Chart I. presents a table of the rise and fall of the leucocytosis, pulse and temperature (recorded at the time of the leucocyte count in each instance), time of examination relative to operation, and the character of the operation made in each case of the two series.

In studying this chart the following points are noted:

1. Case VII., Series I. (appendectomy; active inflammation not present). On the second day after operation two counts were made, four hours apart, as, owing to chill and rise in temperature, with the appearance of icterus, sepsis was feared, which the leucocyte count excluded.

2. Case V., Series II. (removal of gallstones). This case was recorded for fourteen days after operation, owing to continued leucocytosis, the explanation of which lay in an obstinate and persistent diarrhœa, with some bloody discharge from the bowels and considerable secondary anæmia, as shown by the final examination.

3. Same case as above. This count was made three days prior to operation, and, owing to my absence from town, observations on the second and third days after operation were not obtained.

4. Case VII., Series II. (laparotomy for abdominal tuberculosis). This case died from shock of multiple perforation of the gut sixty-six hours after operation and twelve hours after the last leucocyte count, which was made evidently about two hours *after* perforation occurred. Necropsy showed an extensive "dry gangrenous" condition of a large part of the intestinal wall.

This is the one exception to the generally fair health in all of the cases recorded.

From the foregoing chart a supplementary table of averages is compiled, representing graphically the mutual relation of pulse, temperature, and leucocyte variation. (Chart II.)

This relation, as may be seen by reference to Chart I., is not constant; indeed, except in a general way, the pulse, temperature, and leucocytosis appear to bear no direct relationship to each other in aseptic wound-repair.

A few salient points should be noted from these tables:

(*a*) Of the seventeen cases recorded, maximum leucocytosis was reached in from six to twelve hours after operation in nine, in from twenty-four to thirty hours after operation in six, and in from forty-eight to fifty-four hours after operation in one.

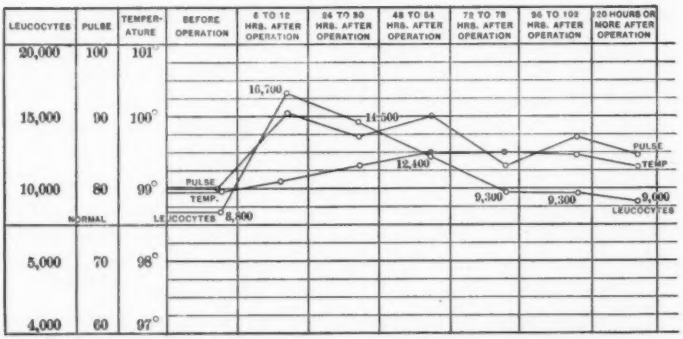
CHART I. (SERIES I. AND II.)—TABLE OF LEUCOCYTOSIS, PULSE, TEMPERATURE, ETC., IN SEVENTEEN CASES.

Case.	6 to 24 hours prior to operation.	6 to 12 hours after operation.	24 to 30 hours after operation.	48 to 54 hours after operation.	72 to 78 hours after operation.	96 to 102 hours after operation.	120 or more hours after operation.	Remarks.
I.	14,700 P. 90 T. 99	18,800 P. 88 T. 100. 2	20,000 P. 84 T. 99. 8	12,100 P. 80 T. 100. 4	13,000 P. 96 T. 99. 6	7,800 P. 94 T. 99. 4	9,300 P. 88 T. 99. 4	Oöphorectomy.
II.	12,500 P. 86 T. 99.4	24,000 P. 84 T. 99. 2	13,400 P. 86 T. 100. 2	9.400 P. 90 T. 100. 4	7,500 P. 84 T. 100. 2	7,200 P. 88 T. 100	.....	Oöphorectomy (malignant).
III.	5,000 P. 88 T. 99	10,000 P. 84 T. 99. 2	11,000 P. 78 T. 99	8,000 P. 68 T. 100. 2	5,000 P. 64 T. 99. 6	.....	.....	Ventrofixation.
IV.	5,000 P. 84 T. 98.5	15,000 P. 100 T. 98	15,600 P. 102 T. 99. 4	18,000 P. 94 T. 99. 2	10,000 P. 90 T. 99. 2	10,000 P. 100 T. 99. 6	5,900 P. 92 T. 100. 2	Ventrofixation.
V.	9,000 P. 76 T. 98.5	17,000 P. 90 T. 99. 4	11,000 P. 94 T. 99. 2	11,000 P. 90 T. 98. 5	9,400 P. 90 T. 98. 5	8,100 P. 96 T. 99	.....	Oöphorectomy.
VI.	12,200 P. 80 T. 99.8	13,800 P. 80 T. 99	6,600 P. 84 T. 99. 4	13,100 P. 80 T. 99. 8	11,800 P. 76 T. 99. 8	9,000 P. 64 T. 99. 4	.....	Oöphorectomy.
VII.	10,000 P. 60 T. 98.4	18,100 P. 70 T. 98. 8	11,200 P. 60 T. 99	7,000 7.200 P. 80 T. 100. 4	12,000 P. 72 T. 99	10,500 P. 60 T. 99	6,800 P. 60 T. 98. 8	Appendectomy.
VIII.	7,500 P. 70 T. 98	18,800 P. 112 T. 99	16,300 P. 102 T. 99. 6	11,900 P. 90 T. 99. 4	6,300 P. 90 T. 99. 6	.....	.....	Amputation of breast (malignant).
IX.	3,800 P. 68 T. 99.6	7,800 P. 84 T. 99. 2	10,900 P. 100 T. 99. 2	7,500 P. 92 T. 100	7,500 P. 89 T. 98. 6	2,800 P. 81 T. 99. 4	.....	Cystic tumor.
X.	5,300 P. 102 T. 100	10,900 P. 88 T. 98. 4	11,200 P. 80 T. 99. 4	.....	6,600 P. 74 T. 99. 8	.....	.....	Cystic tumor.
I.	5,900 P. 64 T. 99.4	12,800 P. 80 T. 98	20,900 P. 96 T. 99. 8	15,600 P. 76 T. 98	10,600 P. 72 T. 99	7,500 P. 72 T. 100	5,900 P. 72 T. 99. 6	Removal of tubes and ovaries.
II.	16,500 P. 94 T. 99.4	26,300 P. 100 T. 99. 8	18,800 P. 86 T. 100	20,000 P. 90 T. 99. 8	10,000 P. 90 T. 100. 6	13,100 P. 110 T. 100. 6	14,300 P. 90 T. 100	Ventrofixation and adhesions.
III.	6,300 P. 64 T. 97.8	7,200 P. 100 T. 100	4,400 P. 82 T. 98. 8	6,000 P. 74 T. 98. 5	5,000 P. 80 T. 99	6,900 P. 72 T. 98. 6	.....	Oöphorectomy.
IV.	7,500 P. ... T. ...	17,800 P. 82 T. ...	11,900 P. 72 T. 98. 8	.....	10,700 P. 86 T. 100. 1	6,600 P. 76 T. 99. 1	.....	Oöphorectomy.
V.	5,000 P. 70 T. 98	14,000 P. 96 T. 98. 8	12,500 P. 72 T. 98. 8	.....	.....	18,500 P. 108 T. 98. 6	16,000 P. 114 T. 99. 4	Gallstones.
VI.	10,000 P. 68 T. 98.2	.....	21,500 P. 80 T. 98. 6	16,000 P. 98 T 99. 6	13,100 P. 90 T. 99. 6	12,800 P. 98 T. 99	10,000 P. 90 T. 98. 6	Ventral hernia.
VII.	14,000 P. 122 T. 100. 2	25,000 P. 114 T. 100. 6	26,000 P. 106 T. 99. 4	19,000 P. 160 T. sub.	.....	.....	.....	Laparotomy for abdominal tuberculosis.

(*b*) Exclusive of Case VII., Series II.—which from its nature should obviously be omitted from present consideration—the five highest leucocyte counts recorded occurred as follows: Case II., Series II., 26,300, six hours after operation—ventrofixation; Case II., Series I., 24,000, six hours after operation—oöphorectomy; Case VI., Series II., 21,500, twenty-four hours after operation—ventral hernia; Case I., Series II., 20,900, twenty-four hours after operation—oöphorectomy, etc.; Case I., Series I., 20,000, twenty-four hours after operation—oöphorectomy. Excepting in these cases no leucocytosis of 20,000 to 1 cubic millimetre or more was observed.

(*c*) One other of these cases requires special mention—Case IX., Series I. It will be observed that the leucocyte count before operation in this case indicated a condition of leucopænia, being 3800 (considerable chlorosis also existed in this case, the color index being 0.50), and on the fourth day after operation it fell to 2800. The maximum leucocytosis in this case occurred about thirty hours after operation, and amounted to 10,900, which would be equivalent to 15,000 in one whose blood was normal to start with.

CHART II.



Average pulse, temperature, and leucocytosis in seventeen cases.

(*d*) The least variation in the leucocyte count occurred in Case III., Series II., which was an oöphorectomy. The nature of the operation in itself, therefore, seems to have little effect upon the degree of leucocytosis.

From Chart II.—which represents graphically the *average* pulse, temperature, and leucocytosis in the seventeen cases—it will be seen that the leucocytosis (taking the average) rises to its maximum in from six to twelve hours after operation, from which point it steadily declines to normal, which may be said to be reached about one hundred and twenty hours after operation. That there may be many exceptions to this average, however, without signifying sepsis is a fact demonstrated in the tables of Chart I.

CHART III. (SERIES II.)—PER CENT. AND ACTUAL NUMBER PER C.MM. OF RESPECTIVE VARIETIES OF LEUCOCYTES BEFORE AND AFTER OPERATION.

Case.	Time.	Small lympho-cytes.	Large lympho-cytes.	Poly-morpho-nuclear neutro-philes.	Eosino-philes.	Transi-tional forms.	Myelo-cytes.	Total.
I.	12 hours prior to operation	31 % 1829	12 % 708	54 % 3186	Small fract. %	3 % 177	.....	5,900
I.	30 hours after operation	2 % 418	11 % 2299	84 % 17556	None	3 % 627	.....	20,900
I.	126 hours after operation	9 % 531	12 % 708	75¼ % 4454+	⅛ % 29+	3 % 177	.....	5,900
II.	18 hours prior to operation	10½ % 1732+	5 % 825	82 % 13530	.....	2½ % 412+	.....	16,500
II.	24 hours after operation	9 % 1692	7½ % 1410	81 % 15228	.....	2 % 376	.....	18,800
II.	168 hours after operation	22 % 3366	11¼ % 1759+	61½ % 9409+	3½ % 535+	1 % 153	½ % 76+	15,300
III.	18 hours prior to operation	35 % 2205	15 % 945	45 % 2835	1 % 63	4 % 252	.....	6,300
III.	24 hours after operation	7 % 504	6 % 432	84 % 6048	.....	3 % 216	.....	7,200
III.	120 hours after operation	37 % 2553	6 % 414	53 % 3657	1 % 69	3 % 207	.....	6,900
IV.	12 hours prior to operation	43 % 3225	5 % 375	49 % 3675	1 % 75	2 % 150	.....	7,500
IV.	30 hours after operation	9 % 1071	4 % 476	83 % 9877	.....	4 % 476	.....	11,900
IV.	126 hours after operation	28 % 1848	7 % 462	60 % 3960	1 % 66	3 % 189	1 % 66	6,600
V.[1]	3 days prior to operation	25 % 1250	10 % 500	62 % 3100	.....	3 % 150	.....	5,000
V.	6 days after operation	8 % 1000	10 % 1250	78 % 9750	1 % 125	3 % 375	.....	12,500
V.	14 days after operation	18½ % 2886	8 % 1248	68½ % 10686	2 % 312	2½ % 390	½ % 78	15,600
VI.	12 hours prior to operation	10 % 1000	16 % 1600	68 % 6800	3 % 300	3 % 300	.....	10,000
VI.	30 hours after operation	7 % 1505	5 % 1075	86 % 18490	.....	2 % 430	.....	21,500
VI.	150 hours after operation	13 % 780	10 % 600	75 % 4800	1 % 60	1 % 60	.....	6,000
VII.[2]	60 hours prior to operation	19 % 2660	11½ % 1610	68 % 9520	½ % 70	1 % 140	.....	14,000
VII.	30 hours after operation	7½ % 1950	6½ % 1690	85 % 22100	.....	1 % 260	.....	26,000

Chart III. is a table of the differential computation of leucocytes, in percentages and actual number per cubic millimetre, made shortly before operation, again twenty-four to thirty hours after operation (except in one instance), and lastly, one hundred and twenty or more hours after operation (also excepting one instance) in the seven cases of the second series.

[1] In this case an accident to the cover-glass preparations prevented a differential computation on the first day after operation. One was made, therefore, on the sixth day when leucocytosis had declined to about the same degree as that of the first day after operation. Leucocytosis persisted at the fourteenth day, owing to diarrhœa and secondary anæmia. This experiment is therefore vitiated.

[2] This is the case which died from shock sixty-six hours after operation, making the final examination impossible.

The chief object here is to demonstrate again what has so frequently been done before, namely, that the principal change in the colorless elements of the blood in leucocytosis occurs in the neutrophiles, which are increased out of all proportion to the other leucocytes; indeed, in the instance of the small lymphocytes in my seven cases the second examination made during the maximum leucocytosis showed actual diminution in number per cubic millimetre in every case but one (Case VI., Chart III.).

The large lymphocytes are found, at the same time, slightly increased in actual number per cubic millimetre except in two cases (Cases III. and VI., Chart III.).

Transitional forms, also, at the same time are increased to some extent except in one case (Case III., Chart III.).

In the case of the polymorphonuclear neutrophiles, however, there are no exceptions, and the increase in actual number is so considerable as to be readily apparent, while in relative number their percentage rises sharply in all but one case (Case II., Chart III.), where it remains nearly stationary, owing to the existence of a marked leucocytosis prior to operation, with comparatively little variation in this respect during the whole course of observations. (See Case II., Series II., Chart I.)

In the first series of examinations, as before stated, two counts of the red corpuscles were made in eight cases—the first count shortly prior to operation, and the second from twenty-four to thirty hours after operation. I expected to find a diminution in erythrocytes following operation, especially in cases where any considerable blood was lost. The contrary, however, obtained. My attention was about this time called to an excerpt from an article by Ermanno Pinzani,[1] who found the red cells and hæmoglobin increased following oöphorectomy, and evidently believed the increase to be real.

The results in the eight cases of my first series were as follows:

Case III.—The red corpuscles increase, in the second examination, more than 500,000 over the count in the first examination; hæmoglobin remaining the same, thus lowering the "color index."

Case IV.—The numerical increase in the second over the first examination is 1,400,000, while the hæmoglobin advances from 70 per cent. to 90 per cent.

Case V.—The cellular increase is 1,000,000; hæmoglobin, 5 per cent.

Case VI.—In this case the increase is insignificant.

Case VII.—The numerical increase is 2,100,000, and hæmoglobin, 5 per cent.

Case VIII.—The increase is again insignificant, being only 100,000, hæmoglobin remaining unchanged.

[1] American Journal of Obstetrics, April, 1899.

Case IX.—The numerical advance is 300,000; hæmoglobin, 5 per cent.

Case X.—The cell increase is 500,000, while the hæmoglobin advances 15 per cent.

On account of these rather unexpected findings it was thought best to carry the observations a little further in the second series. A third examination was, therefore, made in the latter series, one hundred and twenty hours or more after operation, to determine whether this increase in erythrocytes was sustained. At the same time cover-glass preparations from each examination were stained and a systematic search made for nucleated red cells. It was believed that if the advance in red corpuscles were real, and not merely a phenomenon of transient character occurring in the peripheral blood, there would be a noticeable increase of nucleated red cells in the circulating blood, such as obtains in any blood regenerative process, notably in the secondary anæmia following severe hemorrhage.

The results of these observations in the second series are shown in the tables of Chart IV. It will be seen that while in every instance the second examination shows an increase in erythrocytes over the first of from 100,000 to 1,000,000, the third examination shows a return of the red cells to practically the number counted at the first examination. The same is true of the hæmoglobin to all practical purposes.

The rule adopted in regard to the nucleated red cells was to record all observed while counting 300 leucocytes, taking a different cover-glass preparation for each 100 or 150 leucocytes counted, to avoid errors arising from faulty distribution of blood.

Only one case (Case I., Chart IV.) shows any practical increase in the number of nucleated erythrocytes. It is significant that this is the case which shows the greatest and most persistent increase in red cells of any of the series.

In view of the findings in this series it must, I think, be admitted that the apparent increase in the red cells following operation is not real—*i. e.*, due to the excitation of the hæmogenetic function, but rather is due either to the temporary abstraction of the fluid constituents from the blood or merely a phenomenon of the peripheral blood and of transient character.

To a certain extent the leucocytosis might be explained in the same way; but after all allowance is made for such possible error there would still be left no inconsiderable leucocytosis, which is further proved real by the changes in relation between the various forms which have been recorded in Chart III.

It must further be admitted that chloroform anæsthesia (which was employed in all of these cases, sometimes alone, and again only at the start and followed by ether) may play some part in the leucocytosis

following operation; for I have had the opportunity of observing that chloroform anæsthesia alone is capable of producing a leucocytosis in the peripheral blood, which, however, is scarcely more persistent than that produced by the digestion of a hearty meal, and consequently, of course, need not be seriously considered in this connection.

CHART IV. (SERIES II.)—TABLE OF ERYTHROCYTES, HÆMOGLOBIN AND NUCLEATED RED CELLS BEFORE AND AFTER OPERATION.

Case.	Time.	Erythrocytes.	Hæmoglobin (v. Fleischl).	Color index.	Number of nucleated red cells per 300 leucocytes.
I.	12 hours prior to operation	3,800,000	50 %	0.66	One.
I.	30 hours after operation	4,800,000	65 %	0.68	Four.
I.	130 " " "	4,600,000	60 %	0.65	None.
II.	18 hours prior to operation	4,300,000	85 %	1.00	One.
II.	24 hours after operation	4,600,000	92 %	1.00	None.
II.[1]	168 " " "	.....	.....	.....	"
III.	18 hours prior to operation	3,800,000	82 %	1.08	"
III.	24 hours after operation	4,700,000	85 %	0.90	Two.
III.	120 " " "	3,900,000	85 %	1.09	None.
IV.	12 hours prior to operation	3,400,000	80 %	1.18	One.
IV.	30 hours after operation	3,900,000	90 %	1.15	None.
IV.	126 " " "	3,100,000	70 %	1.13	"
V.	3 days prior to operation	3,600,000	70 %	1.00	"
V.	24 hours after operation	3,800,000	75 %	1.00	"
V.[2]	11 days " "	2,400,000	55 %	1.14	"
VI.	12 hours prior to operation	3,400,000	80 %	1.18	"
VI.	30 hours after operation	3,500,000	80 %	1.14	"
VI.	150 " " "	3,000,000	80 %	1.33	"
VII.	2 days prior to operation	3,100,000	50 %	0.80	"
VII.	30 hours after operation	3,900,000	65 %	0.83	"

The conclusions which I think may be safely drawn from the foregoing are:

1. An increase of from 5000 to 10,000 leucocytes per cubic millimetre following operation in from six to thirty-six or even forty-eight

[1] The record of this examination for estimation of red cells and hæmoglobin has been lost.
[2] This is the case before spoken of, in which a persistent diarrhœa followed operation, causing secondary anæmia.

hours is a normal post-operative condition, provided it be not sustained.

2. Probably the maximum leucocytosis in the majority of cases occurs within the first twelve hours after operation, and is very transient.

3. The leucocytosis in the normal reparative process bears but slight relation to the pulse and temperature.

4. A post-operative leucocytosis of 10,000 or more *above the individual normal* sustained for more than a few hours may be looked upon with suspicion.

5. The apparent increase in number of erythrocytes following operation is not caused by an actual increase of red cells in the circulating blood.

To Drs. Eugene Boise and Richard R. Smith, of Butterworth Hospital, and Drs. Schuyler Graves and O. E. Herrick, of the Union Benevolent Association Hospital, I am indebted for the opportunity of making these observations, every courtesy possible being extended me by all of these gentlemen during their respective services.

ENTRANCE OF AIR INTO THE VEINS, AND ITS TREATMENT.

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Although the dangers accompanying sudden entrance of air into the venous circulation were recognized early in the eighteenth century by such observers as Redi, Valsalva, and Morgagni, it remained for Beauchesne, in 1818, to first report a death, verified by autopsy, due to entrance of air into the jugular vein during an operation for the removal of a tumor from the neck.

The discussion following Beauchesne's report has been carried on during the past century by such clinicians and experimentalists as Barlow, Mirault, Delaporte, Ammussat, Cormack, Wattman, and others—during the early part of the nineteenth century—and later by Senn, Hare, Treves, Porter, Hektoen, Draper, and Davidson.

The conclusions which have been reached are various. On the one hand, we have heard warnings against its dangers; others have endeavored to dispel the dread of this accident existing among surgeons. Some have taught that the merest bubble of air entering the veins may cause alarming symptoms, possibly death; while others have

claimed that large quantities of air may be injected into the veins near the heart with no ill effects save a slight, temporary fall in the blood-pressure. Even among those who have believed in the dangers attending the accident, opinions have differed as to the direct cause of death.

Treves believed that death was due to arrest of pulmonary circulation, the air in the right heart being mixed with blood, and preventing action of the tricuspid and pulmonary valves, but little blood reaching the left ventricle, and anæmia of the brain following.

Cormack thought that death was due to gaseous distention of the right heart.

Bell asserted that the cause of death lay in the transference of air to the base of the brain, with consequent respiratory paralysis.

Virchow claimed that complete air embolus of the pulmonary artery, with consequent loss of blood to the coronary circulation and arrest of the heart in diastole, was the immediate cause of death.

In citing these cases I have by no means exhausted the differences in opinion which have existed during the past century among eminent observers.

While the cause of death has been a subject of much controversy, the fact that death does occur and is due directly to the entrance of air into the veins is acknowledged by surgeons generally, there being few notable exceptions.

Hare, in 1889, published a paper in the *Therapeutic Gazette* voicing views on this subject at variance with the popular idea. In comparing his paper with that of Senn's, published four years previously, we must be impressed with the fact that in experimental work personal equation plays no insignificant part. Both are men whose opinions carry weight; and yet we find, on the one hand, Hare, after experimenting with some seventy dogs, asserting that even large quantities of air do little harm, while small amounts, varying from 20 c.c. to 40 c.c., injected into the jugular vein cause little more than slight transitory fall in blood-pressure; on the other hand, Senn found that small quantities of air when injected into the jugular vein proved fatal in a large percentage of his dogs.

While this accident occurs more commonly during operations in the neck and axillary regions, the veins in other parts of the body are by no means exempt.

Davidson reports the case of a Hindoo woman who died suddenly, after a normal labor, and in whom the autopsy revealed dilated uterine veins, congested lungs, and a right ventricle filled with mixed air and blood.

So, also, Wyman reports a death following rapidly the aspiration of a pelvic tumor, where the autopsy showed a right heart dilated with air, while neither ventricle contained any blood.

Volkmann reported a death due to entrance of air while aspirating a longitudinal sinus.

Bergmann describes the case of a man whose occiput was injured; the man died in four hours. On post-mortem examination both lungs were found œdematous, and there was a very extensive form of air embolism, the air probably entering through the injured torcular.

A death has been reported from entrance of air into the sinuses of the dura mater. However, so much more frequently has this accident occurred during operations in the region of the axilla and neck that this has come to be known as the "danger zone."

Senn says: "Clinical observation and experimental research have established the fact that the venous circulation is directly influenced by respiration within a certain area, and that inspiration of air, in the majority of cases, takes place in the veins thus affected, constituting the justly much-dreaded 'danger zone.'"

It is in these veins near the heart that the so-called suction force is exerted. When a vein is completely severed and its lumen closed by collapse of its walls the danger is practically *nil*. Partial division or nicking of the vessel constitutes the condition favorable to the drawing of air into the vein.

The peculiar relation of the external jugular vein to the cervical fascia and of the axillary vein to the costo-coracoid fascia renders the lumen of these vessels patulous even when cut through. This is also true of veins whose walls have been diseased, thickened by calcareous deposits, or in such conditions as periphlebitis, where the surrounding inflammatory tissue is intimately connected with the walls of the veins. Thus the factors in this region upon which this accident depends are two—patulous veins and suction force exerted during inspiration.

The accident has occurred with greatest frequency during removal of tumors from the neck and axilla, amputation at the shoulder-joint, resection of the scapula, ligation of the subclavian, and venesection of the jugular vein; but it may occur in the most trivial operation in this region, and more frequently in the cervical veins than in the axillary.

The symptoms attending this accident as described by various surgeons and coinciding with my own observations in experiments on dogs are: A hissing noise accompanies the entrance of air into the vein; the blood-pressure immediately falls; the heart—at first tumultuous, with a churning sound plainly heard over the præcordial area, synchronous with the heart action—becomes rapidly weak and irregular; convulsive twitchings of the entire body occur; at this time (and the preceding symptoms have occupied scarcely thirty seconds) the pulse has disappeared, the heart has ceased except for an occasional impulse, the breathing becomes labored and rapid, and after a few violent

inspiratory efforts the animal dies. The picture is one of cardiac failure, with secondary respiratory paralysis due to anæmia of the brain centres.

While working with Dr. Kemp in the Columbia Physiological Laboratory, two years ago, to determine the effects on blood-pressure of infusion with normal salt solution introduced at temperatures ranging from 105° to 120° F. the following accident occurred:

A moderate-sized dog was given a dgm. of morphine at 10 A.M.; at 10.45 A.M. ether was administered. When the animal was completely anæsthetized a tracheotomy was performed for convenience in administering the ether during the further course of the experiment.

The right carotid was then opened, and into it was inserted a manometer tube, which in turn was attached to the kymograph, so that any changes in blood-pressure occurring during the experiment might readily be noted.

The right femoral artery was exposed, and into it a canula was tied. As a final procedure the femoral vein was opened, and through the canula placed in its lumen we expected to perform our infusion. The saline infusion was then prepared at a temperature of 105° F. and placed in the douche bag. The kymograph was set in motion and the normal blood-pressure noted—*i. e.*, 182 mm. We now removed through the canula placed in the femoral artery 100 c.c. of blood; the pressure slowly fell during the bleeding to 110 mm. After waiting two minutes for spontaneous recovery in pressure, which invariably occurs after bleeding, we prepared to start our infusion.

The canula in the femoral vein was carefully filled with salt solution, so that no air might enter when the infusion was started. Through some misunderstanding the same precaution was not exercised on the tube of the douche bag, consequently it was filled with air between the clip and the distal end; so when it was attached to the canula in the vein and the clip opened, a column of air 3 feet long and 6 mm. in diameter was forced into the vein by the pressure of the fluid in the bag; about 12 c.c. of air must have been injected in this way. The air was plainly seen rushing through the glass canula. The blood-pressure fell in a few seconds to near zero. (See Fig. 1.) The churning sound observed in these cases was distinctly heard; the heart's action became at first tumultuous, and then rapid and weak; convulsive muscular twitching began; respirations were labored and inspirations deep and gasping; death seemed certain. Instead of removing the canula and stopping the infusion the saline was immediately increased in temperature to 120° F., and artificial respiration by means of Hoyt's artificial respiration apparatus was begun. Slowly the action of the heart returned; a slight tracing reappeared on the drum, and the blood-pressure slowly arose. The infusion was continued, being given very slowly; artificial respiration was fixed at 20 per minute; in fifteen minutes the heart had regained its expansile force, the tracing on the drum was normal, and respiration was much improved. Accordingly both infusion and artificial respiration were stopped. The blood-pressure remained at 156 mm., and we continued our original intentions on the same dog for the subsequent hour and a half.

This experiment was particularly valuable because of the fact that the entrance of air was entirely accidental, and happened under conditions that might well have occurred clinically. It seemed to show that the entrance of air into a vein under pressure was dangerous and might lead to a fatal termination. It also seemed that in the treatment of this condition direct venous infusion with normal salt solution at a temperature of 115° to 120° F. and artificial respiration were important factors. I was lead by this result to continue the experiments on a series of dogs at the Columbia Physiological Laboratory last winter.

In the first five experiments the following routine was followed: The dog was given morphine in dosage from 5 cgm. to 1 dgm., according to its size; after waiting half an hour the animal was etherized and tracheotomy was performed. The femoral vein was exposed for injection of air and infusion; a canula with a stopcock attachment connecting with the atmospheric air, in order that the syringe might be filled without removal from the vein, was inserted and tied in place, and the carotid artery on the right side was then exposed and connected by means of a manometer tube to the kymograph. I have chosen Experiment 3 as a fair representative of this class.

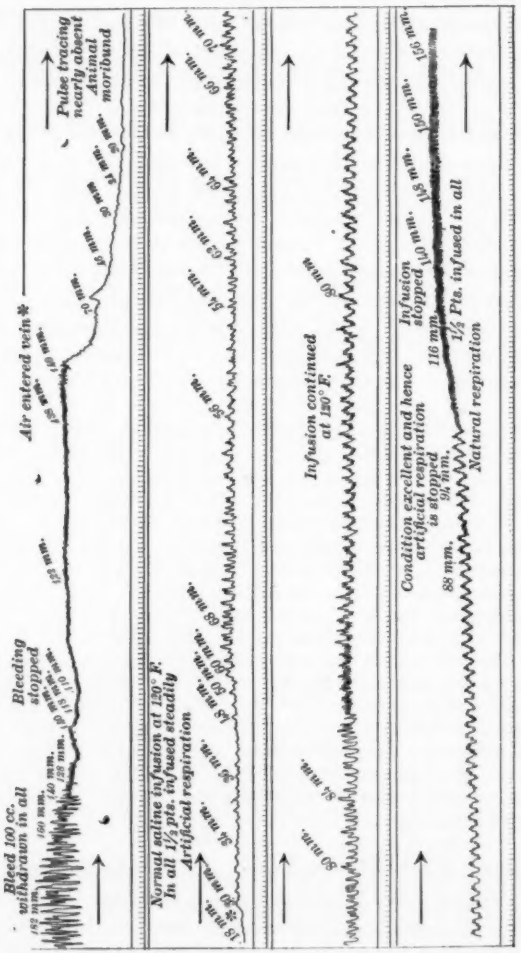
A white setter, weighing forty pounds, was given 1 dgm. of morphine, and 75 c.c. of air was injected into the vein. The pressure dropped from 124 mm. to 8 mm. in a period of time covering fifty seconds. Immediately following the entrance of air the heart became tumultuous; a churning sound was plainly heard in the chest; in a few seconds the heart became weak and irregular, the femoral pulse disappeared, and finally the heart ceased to beat perceptibly. The respirations were not affected until some seconds after the heart began to show the first changes; then they became labored and more rapid. This was followed by the peculiar forced inspirations noted in all these cases, and the respirations did not cease entirely until one minute after the last heart tracing was noted on the drum. An infusion of normal salt solution at a temperature of 120° F. was given, without the slightest result; the dog was dead. The autopsy revealed the right ventricle distended with air; the large venous trunks contained many air bubbles, as did the pulmonary artery. The coronary vessels were found filled with air emboli and the heart was fibrillating; this continued for about fifteen minutes.

In all these dogs the air was injected with considerable force; the maximum amount used in any individual case was 75 c.c. Infusion was resorted to in six dogs, with recovery in two. In one of the dogs which recovered I believe such would have been the case with no treatment; thus there was but one favorable result following treatment by infusion alone. (See Fig. 1—accidental entrance of air during infusion.)

In one dog, after a temporary rise in blood-pressure, too much infusion was allowed to enter the vein, and it died of pulmonary œdema.

In another dog death occurred when artificial respiration was begun after a temporary rise in blood-pressure had been noted following infusion.

FIG. 1.


Bleed 100 cc. withdrawn in all
Bleeding stopped
Air entered vein*
Pulse tracing nearly absent Animal moribund
Normal saline infusion at 120° F. In all 1½ pts. infused steadily
Artificial respiration
Infusion continued at 120° F.
Condition excellent and hence artificial respiration is stopped
Infusion stopped
1½ Pts. infused in all
Natural respiration

On the rest of the dogs experimented upon infusion had no effect whatever. In all the primary effect of air injection was on the heart, the respiratory symptoms being secondary. An autopsy was performed

after each experiment, except Experiment 1, where entrance of air was accidental, and in all the animals the coronary vessels were found full of air emboli and the heart fibrillating; in four of the five the right ventricle was distended with air.

I am inclined to believe that death in these cases was due to one of two possible causes: First, gaseous distention of the right ventricle, with loss of contractile power, failure of both pulmonary and tricuspid valves to act, failure of blood to reach the left ventricle, and consequently anæmia of the brain centres. The second, and it seems to me the more important factor in the death of these animals, was the plugging of the coronary vessels with air emboli. The heart being deprived of its circulation was thrown into fibrillation, and death ensued. This was observed by Cunningham in 1898. Erichsen, Cohnheim, Rechberg, Porter, and others found that by tying off the coronary vessels or injecting them with a preparation of wax, paraffin, and soot, thus completely shutting off the intrinsic circulation of the heart, the heart becomes irregular, the pressure sinks, and the ventricles beat arrythmically; that is, they cease to beat as a unit, but individual muscle fibres continue an inco-ordinate contraction for some time. This is precisely the condition observed in these dogs.

I cannot agree that respiratory failure plays a primary part in the result, as is believed by Hare and others. Hare reports two cases in which he injected the carotid artery; in one 40 c.c. of air was used, and in the other 20 c.c.; each was equally fatal, and respiratory failure was in these cases the primary cause of death, the heart beating some time after respiration ceased. His deductions based on these experiments are as follows:

"Under such circumstances" (air injected into the carotid) "the results are quite different from those seen after opening the jugular vein, but nevertheless totally disprove the theory that it is by cardiac failure that death under such circumstances occurs." This statement holds good only in so far as injection of the arteries with air is concerned, air emboli being carried directly to the base of the brain and the respiratory centres, while, when the venous system is injected the air is carried to the right ventricle, and the resulting mechanical dilatation of that receptacle causes anæmia of the brain centres and secondary respiratory paralysis. If Hare means to apply the results of his experiments with injection of air into the arteries to similar injections of the veins I think his conclusions are obviously erroneous. (See Fig. 5, Experiment 17.) Rapid injection of air was an important factor in obtaining the preceding results, for, as will be seen later, considerable quantities of air may be slowly injected without serious effect.

I was impressed with two facts in my first experiments: First, that entrance of air into the veins is a serious accident, which may well

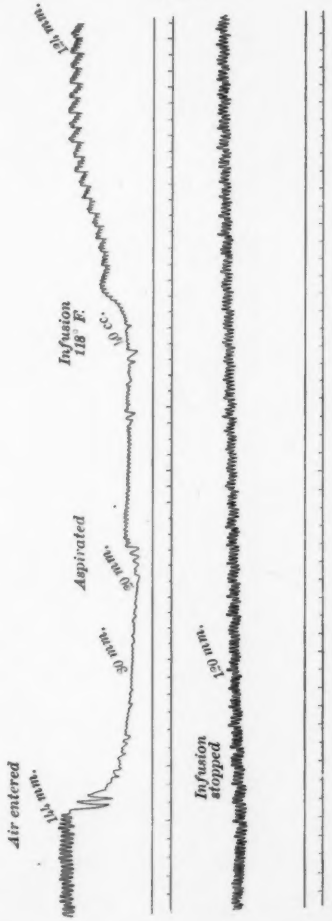
result fatally; and secondly, that when the right heart is distended with air, infusion can be of little service until that air is removed. I believe with Treves that artificial respiration is unnecessary, if not contra-indicated, there being no primary respiratory failure, and the dyspnœa being due not to lack of air, but lack of blood in the lungs.

In 1885 Senn, after injecting air into the veins of dogs, aspirated the right heart by means of a needle about 2 mm. in diameter, forced through the chest wall into the ventricle; 30 per cent. of his dogs recovered after this treatment. It seemed to me that if this were followed by infusion of saline at high temperatures we might expect even a higher percentage of recoveries. Accordingly I experimented on some eleven dogs, with this object in view. The same routine was followed as in the former experiments, with two differences: the external jugular vein was used for our injections of air instead of the femoral, and in the treatment an aspirating needle, 2 mm. in diameter, was forced into the right ventricle, and the air removed by means of Hoyt's vacuum pump before infusion was begun. I shall let Experiments 10, 13, 15, and 17 represent this class.

Experiment 10 (No. 1).—This dog—a heavily built bulldog, weighing 34.5 pounds—was given 1 dgm. of morphine about an hour before the anæsthetic was commenced. As we intended to allow this dog to come out of ether we did not do tracheotomy. As much care as possible was used in asepsis; after anæsthetizing, the carotid and jugular were exposed and treated as in the other experiments. Everything being ready, the kymograph was set in motion and the normal pressure noted; in this case it was 148 mm. After waiting five minutes 75 c.c. of air from an Ultzman syringe was injected into the left jugular vein. The effect was instantaneous; within ten seconds the pressure fell from 140 mm. to 40 mm.; the heart was tumultuous; the churning sound before described was plainly heard; in a few more seconds the pressure had fallen to 18 mm. (which is practically zero, as it will never fall to actual zero, even when the animal is quite dead, until the arterial canula is removed); the heart grew rapidly weak, until all pulse tracings finally disappeared from the drum; the respirations still continued, however, though labored and gasping at this time. We then inserted a needle into the fourth left interspace, about an inch from the sternum; the vacuum apparatus was set in motion and a vacuum created in the bottle; the needle was then thrust obliquely into the right ventricle, and about 150 c.c. of air mixed with blood was drawn off. The pressure arose immediately, the pulse tracings reappeared on the drum, and the cardiac expansions increased; at this point infusion was begun, and about thirteen ounces of normal salt solution at a temperature of 120° F. was allowed to run in very slowly. One minute after this treatment the pressure had risen to 128 mm.; infusion was stopped, the vessels were tied, and the wound was closed with catgut sutures. The dog was then placed in a cage, was wrapped in blankets, and was surrounded by hot-water bottles. The ether was stopped at 1 P.M.; at 2 P.M. the dog came out from under the influence of his anæsthetic; at 5 P.M. the same day he was weak, but able

to stand. At this time half a pint of milk was given him, which he retained. The next day he got "regular diet." During the next five days he continued to do well, took his nourishment eagerly, and, with the exception of a stitch abscess in the femoral incision, was apparently as well as before the operation.

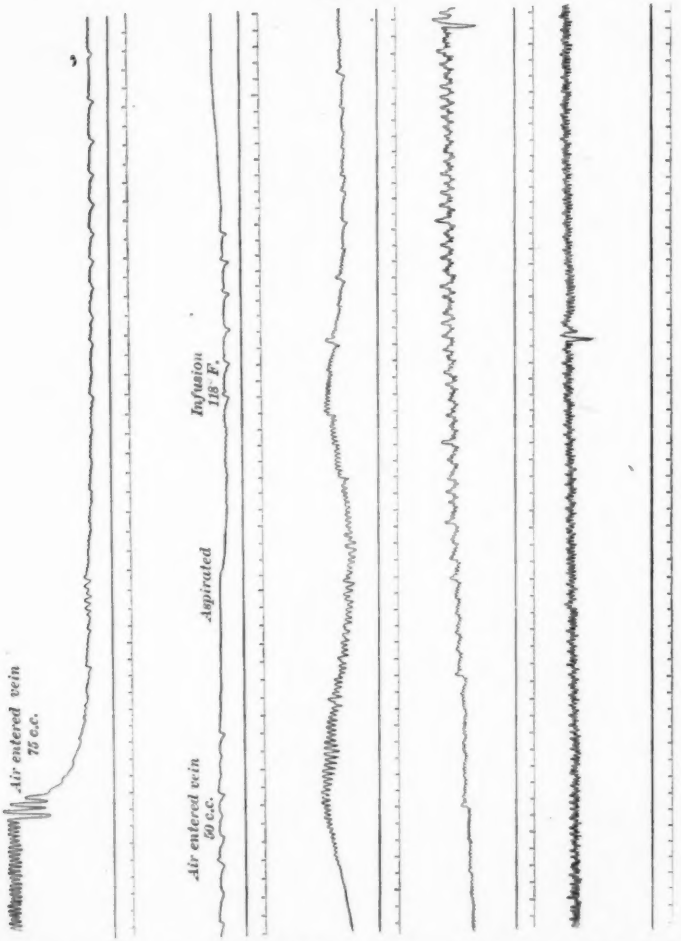
FIG. 2.



No. 2.—On the morning of the sixth day the same dog was again etherized, the opposite jugular was exposed and opened, and 75 c.c. of air was injected. Immediately the same symptoms developed as were present in this dog six days previously. No treatment was resorted to in this case, and the dog died. After waiting for five minutes the chest was

opened, the right ventricle was found distended with air, the auricles were spasmodically contracting, the coronary vessels were filled with air emboli, and the heart was fibrillatiug; this continued for fifteen minutes. The old puncture scar was visible at the base of the right ventricle; there was no blood in the pericardium.

FIG. 3.



Experiment 13.—A dog weighing forty-four pounds was given 8 cgm. of morphine, anæsthetized with ether, and dealt with in the manner above described. Everything being ready, the kymograph was set in motion at 11.38 A.M.; at 11.44 A.M. 75 c.c. of air was

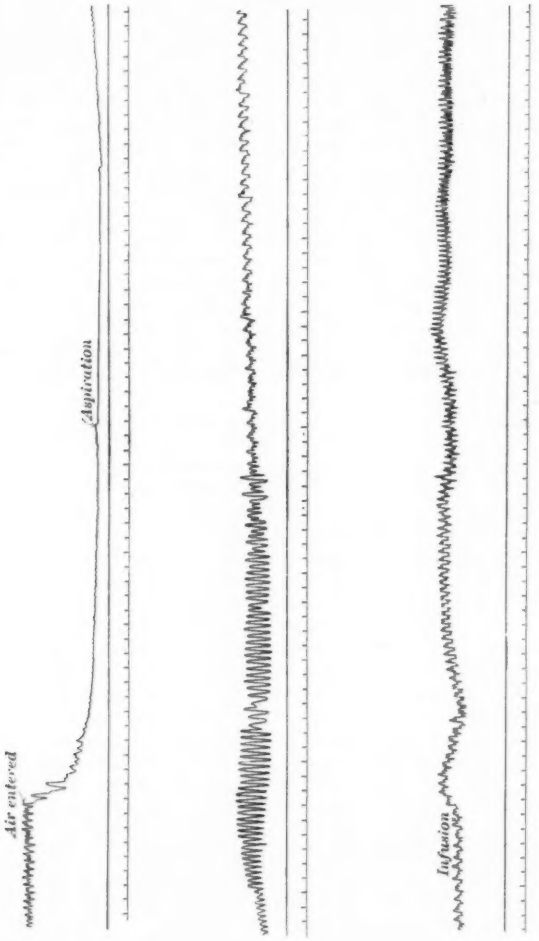
injected into the jugular vein. The blood-pressure immediately fell from 162 mm. to 50 mm.; the heart continued to beat perceptibly, the tracing on the drum showed less expansion, and the pulse was rapid, weak, and irregular. We did not resort to any treatment at this time. After waiting four minutes the pulse began to improve, and the pressure slowly rose until it reached 160 mm., and the dog was in quite as good condition as before the air was forced into the veins; thus in this case 75 c.c. of air had but a transitory effect on the heart. After waiting for twenty minutes (and during that time the heart remained in good condition) we forced 75 c.c. of air into the jugular vein (see Fig. 3); instantly the pressure fell, the femoral pulse disappeared, and the tracing on the drum was barely perceptible; in about two minutes the gasping respiration peculiar to these cases commenced. We then injected 50 c.c. of air; in three minutes after the first injection of air the dog was apparently dead. At this juncture the needle was inserted and the right ventricle was aspirated; in a few seconds a faint impulse was felt against the needle, and this was followed by a feeble tracing on the drum, and respirations began again. An infusion of normal salt solution at a temperature of 120° F. was begun, and the pressure slowly rose until in three minutes it had reached 180 mm., about a pint of salt solution in all having been used. After waiting twenty minutes the dog was killed with chloroform. On opening the chest no air was found in the heart or coronary vessels and no effusion of blood in the pericardium.

Experiment 15.—A fairly large dog weighing thirty-eight pounds was given 9 cgm. of morphine; at 10.30 A.M. anæsthesia with ether was started, and the operation was proceeded with as in the other experiments. At 11.08 A.M. the kymograph was started and a normal tracing obtained; at 11.10 A.M. 50 c.c. of air was injected into the jugular, and this was followed by a slight fall in blood-pressure, which, however, was speedily recovered from, and no treatment was necessary. At 11.15 A.M. 100 c.c. of air was injected; the pressure fell directly from 150 mm. to 12 mm.; no heart-beat was registered; no pulse could be felt; there were several gasping inspirations, and the dog seemed quite dead. After waiting some seconds to make sure that the animal would not recover spontaneously, and still no tracing showing any movement of the heart, the right ventricle was accordingly aspirated, and when a few ounces of mixed air and blood had been drawn off the heart's action could be felt against the needle, and also the tracing and pulse reappeared. In this case no infusion was done; the pressure rose to 150 mm., and the heart's action continued fairly good—a little irregular for a few minutes, and later quite normal. At 11.30 A.M. 100 c.c. of air was injected, causing a fall in blood-pressure, but the heart continued. (See Fig. 4.) At 11.35 A.M. the blood-pressure had risen to 120 mm., and 50 c.c. of additional air was injected. This was sufficient; the pressure fell, and there was neither pulse nor tracing in forty-six seconds. The dog being practically dead, the heart was aspirated, when the pressure rose to 72 mm. in one minute, the pulse being then slightly irregular and feeble. It continued in this manner until 11.40 A.M., when an infusion was done. This was followed by another rise in pressure to 125 mm., an increase in the force of the heart, and a regular pulse. The animal was finally killed by an injection of 100 c.c. of air into the jugular vein.

An autopsy showed the right ventricle filled with air; the coronary vessels contained air emboli; there was no effusion into the pericardium.

Experiment 17 (No. 1).—In this case we determined to do a sterile operation, inject air, aspirate only, and allow the dog to recover if he

FIG. 4.

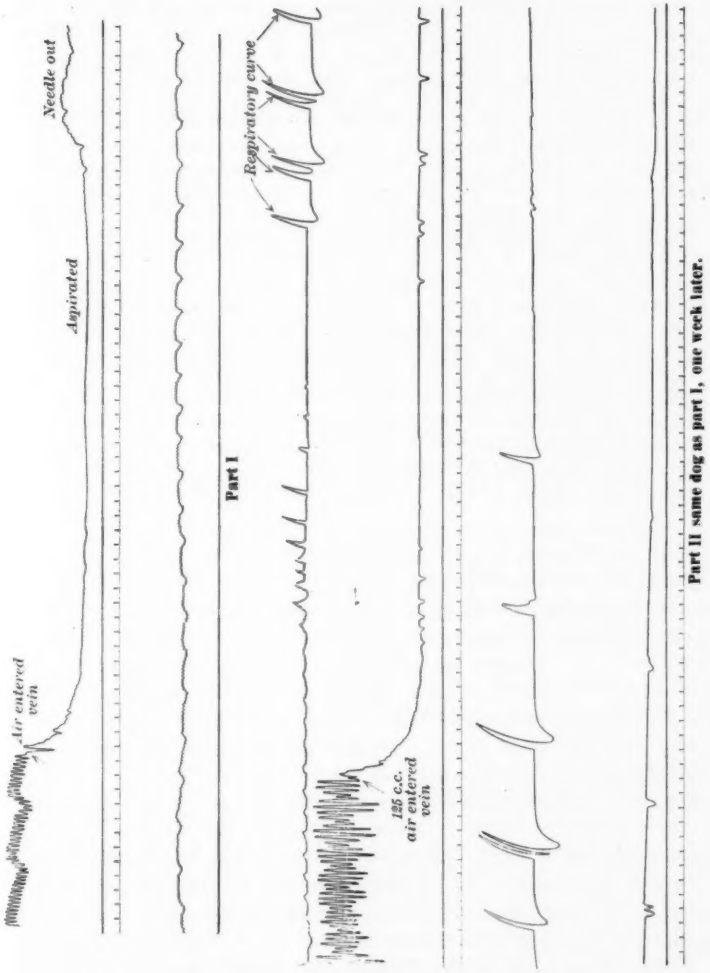


would. The dog in this case was a large, powerful bulldog, weighing forty pounds. He was accordingly given 1 dgm. of morphine at 10.30 A.M., and at 11 A.M. ether anæsthesia was started. The right carotid was opened, a manometer tube was inserted, and also the left jugular

vein was opened. No tracheotomy was done in this case. At 11.34 A.M. 125 c.c. of air was injected into the jugular. (See Fig. 5.) The pressure fell immediately from 140 mm. to 20 mm.; the heart's action

FIG. 5.

FIG. 6.



ceased, no pulse being perceptible, and no pulse tracing was observed for thirty-two seconds; a needle was then introduced into the right ventricle and 150 c.c. of mixed air and blood withdrawn. Almost immediately the pressure began to rise and the heart's action to be

shown on the tracing. In this case the pressure rose very slowly, requiring one-half hour to reach 90 mm., and the expansion of the ventricles was greatly restricted compared with the normal tracing at first obtained. The wounds were sutured and collodion dressing was applied. The dog was placed in a cage and was surrounded by hot-water bottles. He recovered from the anæsthetic about 2 P.M.; when seen at 4 P.M. he was in good condition. The next day he was placed on a "regular diet." The dog recovered strength so rapidly that it was decided to repeat the experiment on the same animal.

Experiment 17 (No. 2).—On the sixth day the dog was given 1 dgm. of morphine, and later was anæsthetized; the opposite vessels to those used in the first case were then exposed and treated as before. In the second operation the respiratory tracing was attached to the kymograph to show the effects of air on the respiration more clearly. When the kymograph was started the large excursion in the heart tracing often noticed after morphine has been given was seen in this case. The heart's action was good; pressure 180 mm.—higher than it had been before the first operation. At 10.55 A.M. the respirations were 20 per minute; pulse 98. At 11.08 A.M. 125 c.c. of air was introduced into the jugular vein; immediately the pressure fell from 180 mm. to 28 mm., and in twenty-four seconds no tracing was perceptible on the drum and no pulse could be felt. There was little change in the respirations for several seconds after this, and they continued for two and one-half minutes after the heart had ceased, deep and gasping, as was beautifully shown on the tracing. (See Fig. 6.) The dog died, and on autopsy the right ventricle, as in other cases, was found dilated with air, and air was found in the coronary vessels.

The great excursion of the respiratory tracing in the preceding experiment demonstrates the deep inspiratory efforts of the animal, caused by anæmia of the brain centres when the cardiac circulation is interfered with. I think that this chart (Fig. 6) clearly shows—Hare's opinion to the contrary notwithstanding—that in the venous injection of air failure of respiration is dependent on cardiac failure.

In eleven experiments (from 7 to 17 inclusive), of which four have been described (*vide supra*), nine dogs recovered after aspiration and infusion, one dog died because the needle used was too small to aspirate the heart, and one dog died in spite of treatment. Two of the animals which finally recovered required large quantities of air (250 to 400 c.c.), injected forcibly, before the pulse tracings on the kymograph entirely disappeared. Three of the dogs which recovered were eventually killed by venous injection of air (see Experiments 10, No. 2, 15, and 17, No. 2), and on autopsy the coronary arteries were found injected with air, the right ventricle was dilated, and the heart was fibrillating. The introduction of the needle into the heart had no ill effects on that organ, and in but one case was any leakage into the pericardium found.

In applying the results of these experiments to the operating-room and the human subject I would divide the treatment into two parts—preventive and radical.

1. *Preventive.* When operating in the region known as the "danger zone" none but the prone position should be permitted, for should one of the veins be injured the semiprone or sitting posture predisposes to entrance of air into the veins. If any dissection is necessary, as is often the case, it should invariably be performed with a blunt instrument, never with a scalpel. Especial care should be used if the operation be one for the removal of cancerous or tuberculous glands, which are always more or less intimately connected with structures adjacent.

2. *Radical.* Should attention be called by the hissing sound accompanying the rush of air into the veins to the injury of a vessel in this region, the finger should be forced into the wound to prevent further entrance of air, the wound cavity should be filled with salt solution, and the chest should be compressed forcibly. If bubbles are seen in the fluid contained in the wound they are due to air coming out. If, however, the heart becomes tumultuous, if the churning sound attracts attention, and if the respirations become labored and the inspirations forced, a needle should be inserted into the fourth left interspace one inch from the left border of the sternum; it should be directed obliquely upward and backward, and the right ventricle will be entered; aspiration should be continued until the blood comes out unmixed with air; then, an assistant having previously prepared a solution of normal salt at a temperature of 115° to 120° F., the patient should be infused until fluid has been introduced equal in quantity to the blood taken from the heart. The most convenient region in which to perform this infusion is the median basilic vein.

To sum up, the following facts seem clear:

1. That entrance of air into the veins, even in small amounts, is to be dreaded, as it may result in death.

2. That death is due to gaseous distention of the right heart or to air emboli in the coronary vessels, and not to primary respiratory paralysis.

3. That combined treatment by aspiration and infusion we may expect to be attended with good results.

In conclusion, I would say that I believe the statement "that large quantities of air may be introduced into the veins without unfavorable result" to be pernicious teaching and not supported by fact.

[I wish to avail myself of this opportunity for thanking Mr. J. T. Hoyt for the very valuable assistance rendered me in his management of the apparatus during the course of these experiments.]

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A CASE OF SUBPECTORAL ABSCESS.

BY GWILYM G. DAVIS, M.D.,
OF PHILADELPHIA.

IN the issue of THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES for November, 1900, Dr. John H. Musser has gone over the literature of this affection, with an abstract of twenty-three cases besides those of his own.

The present case occurred in a man, aged thirty years. He was a worker in the boiler-room of a paper mill. Ten days previous to admission to the Episcopal Hospital, while firing boilers, he became overheated and went outside to cool off. He says this caused him to catch cold, and in a few days he noticed a lump forming on the left side of his chest. He stated that he was subject to colds and attacks of sweating. He was a Pole and spoke no English, and it was difficult to elicit the history.

On admission his temperature was 102° F.; pulse, 118; respiration, 28. On the left side of the chest was a large mass which extended from the nipple below to the clavicle above, and from the edge of the sternum out nearly to the deltoid pectoral groove. It was not red, but was tender and pitted on pressure, and deep fluctuation was plainly evident. The swelling was not pointed but flat, showing that the pus was deep seated. There was no tendency shown anywhere on the surface of pointing or breaking through. The axilla was absolutely healthy, and, as can be seen from the accompanying illustration, the anterior edge of the axillary fold was not encroached on. The depression which normally exists between the inner edges of the deltoid muscle and upper edge of the pectoralis major was perfectly preserved. There were no lesions on the left arm, but on the left hand there were the remains of several old cuts, but nothing recent. He failed to recall any injury to the affected parts or any recent suppurative lesions of the hand. He had an irritative cough with a scanty expectoration of white, frothy sputum. Physical examination of the chest was negative.

An incision was made under the anterior fold of the axilla and several ounces of greenish pus evacuated, a large drainage-tube being introduced. In four days the temperature had fallen to normal, the drainage-tube was removed, and his cough had disappeared. Later he was discharged practically cured.

The pus which was evacuated contained an enormous number of staphylococci, with some streptococci.

In searching for a cause nothing positive could be found. That the coughs to which he was subject originated the trouble is not likely. The cough which he had on admission was evidently secondary to the irritation of the pleura from the inflammation just outside. It ceased as soon as the abscess was evacuated. Had infection followed lung trouble it would have shown itself in the lymphatic glands around the root of the lung. It could only have involved the subpectoral tissues by setting up a localized pleurisy and the infection travelling by direct continuity of tissue. If this had been the origin there would not only have been a previous history or evidence of lung trouble at this point, but the abscess would not have healed so rapidly after being opened. In localized empyemas which point externally healing is usually a very slow process, a sinus remaining for a long time.

If the origin of the infection is sought in the hand, here again we find the evidence insufficient; not only were the cuts slight, but they had evidently been received a long time previously and had given rise to no inflammatory troubles. Infection in such cases shows itself in an involvement of the glands at the inner side of the elbow or the glands in the axilla which lie along the axillary vessels. These were absolutely not involved. A possible explanation might be found in his occupation. Being a fireman and engaged in shovelling coal, it is possible that he may have strained some of the deep fibres of the pectoralis major muscle. The serous effusion following might have become infected through the blood. Calling it idiopathic seems to be begging the question. None of these explanations being satisfactory, we must

simply admit that we do not know the manner and origin of the infection.

An interesting feature of the case is the fact that the abscess showed no signs of pointing, and the question suggests itself as to what would have been the course of events had it been allowed to go on. The pus would not be likely to extend upward on account of the clavicle, and the costo-coracoid membrane.

It might have burrowed inward to the anterior mediastinum or involved the pleura, or, as is most likely, it would have pushed its way toward the axilla and opened along the anterior edge of the axillary folds. If, however, it passed behind instead of in front of the pectoralis minor muscle it would then have bulged into the axilla and showed itself somewhat more posteriorly.

In view of the thickness of the tissues covering the abscess and the lack of any evidences of their implication in the inflammatory process, it is not likely that the pus would have found an exit superficially through the skin.

ANOMALOUS POSITION OF THE COMMON CAROTID, VISIBLE IN THE PHARYNX.

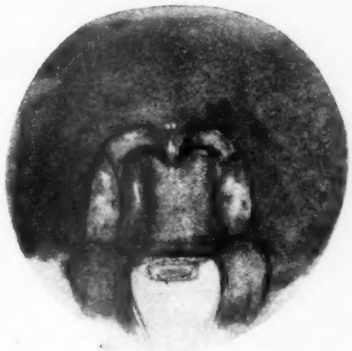
By George B. Wood, M.D.,
of Philadelphia.

Although generally considered of not much importance, anomalies of the carotid artery do occasionally present peculiarities which, under certain diseased conditions, would lead to most unfortunate accidents, and, therefore, I desire to report the following case:

E. M., aged five years, born in Philadelphia, was brought by her mother, because of nasal discharge, to Dr. Freeman's clinic at the Children's Hospital. The child was well nourished, more than usually bright, and seemed perfectly well except for her nasal condition, and apparently possessed no outward abnormality nor deformity. Her catarrhal condition dated from a cold of the previous winter. She sleeps with the mouth closed and has no symptoms of nasal obstruction. The only serious sickness which the child has had was considerable gastro-intestinal trouble when an infant only a month or so old.

Examination. There is practically no enlargement of the cervical lymphatic glands, though one very small nodule can be felt at the angle of the jaw on the left side. On the same side the pulse of the anterior carotid artery is easily felt just behind the angle of the lower jaw, and can be traced downward to the upper border of the thyroid cartilage. On the right side there is no pulsation behind the angle of the jaw, the carotid pulse being first perceived at a point about a quarter of an inch above the upper edge of the thyroid cartilage. From thence downward

the beating of the right carotid can be readily traced. The left nasal fossa is very free, though containing considerable thick mucopurulent secretion, especially along the floor. The septum is fairly straight, and the turbinals smaller than normal. The right fossa is also free, and contains considerable thick, almost crust-like exudate. The pharyngeal vault is freely visible through both fossæ; and the pharyngeal tonsil seems small and not obstructing. The teeth are in perfect condition, and the palatal arch about normal. The fauces are not congested, and the faucial tonsils only slightly enlarged. On the right side of the pharynx, projecting at times almost to the median line, is a large pulsating bloodvessel, about the size of a lead-pencil. By forcibly depressing the tongue, this bloodvessel can be seen running from below upward and slightly inward until it reaches a point about opposite the uvula, whence it takes a course slightly outward and upward. No posterior rhinoscopic view is possible on account of the irritability of the throat. The lingual tonsil and larynx are normal.



Showing the position of the pulsating vessel (×) on the right side of the posterior pharyngeal wall.

The diagnosis of the nasal condition is incipient atrophic rhinitis, but the condition which interests us at present is the arterial anomaly. It is impossible, without a post-mortem examination or dissection, to state exactly what artery is involved and how the blood supply to the throat and nose is changed by the anomalous condition. There are two probabilities: either an enlargement of the internal carotid artery, with absence of the external carotid artery; or an enlargement of the ascending pharyngeal, with absence of the external carotid. The presence of the temporal pulse on the right side shows that at least some of the normal branches of the external carotid are in existence, and that therefore there must be some sufficiently large source which is so located as to be able to give off these branches. On page 502 of Morris' *Anatomy*, under variations of the external carotid artery, we notice the following: "A. It may be absent, the branches usually derived from it

coming off from the upward continuation of the common trunk." Also in Quain's *Anatomy*, "absence of the external carotid artery has been met with in some rare cases, the several branches arising at intervals from a single trunk which represented the internal and common carotids." In Deaver's *Anatomy*, "the external carotid artery may be absent, the branches of that artery arising from the common carotid artery, which continues upward as the internal carotid artery."

From this it appears that a continuation upward from a common carotid trunk may readily account for the findings in our present case. From the position of the bloodvessels, however, it would seem at first sight that the artery involved was an anomalous ascending pharyngeal. The text-books on general anatomy do not mention among the variations of the ascending pharyngeal artery any anomaly of sufficient size to give off the normal branches of the external carotid. Conditions closely resembling the case in question have been described in some of the text-books on diseases of the nose and throat, the descriptions ascribing the anomalous artery to the ascending pharyngeal. On page 488, of Burnett, Ingals, and Newcomb, we find the following: "An abnormal vascular condition not infrequently seen is that of unusually large and pulsating vessels on the posterior and lateral walls of the pharynx. The vessel usually affected is the ascending pharyngeal artery, as determined by its position on the superior constrictor and its vertical direction." Kyle, in his book, on page 479, says: "The branches of the ascending pharyngeal may be unusually large, or the ascending pharyngeal artery itself show distinctly in the wall of the pharynx."

It is not a very uncommon thing in atrophic throats to see the pulsations of a small artery (ascending pharyngeal) on both sides of the median line. The artery in these cases is scarcely larger than one-sixteenth of an inch in diameter, and can scarcely be mistaken for such a condition as is seen in our present case. Further, the more clinical though specialized works, because of their treating the question from a clinical aspect, must of necessity do more or less guessing as to the exact condition of affairs, except, perhaps, where they have obtained their knowledge from some work on anatomy. Therefore, I believe, that while keeping in mind the limits of our present knowledge, the most plausible theory as to the condition of the parts in the case just reported would be to consider the anomalous artery an upward continuation of the common carotid trunk.

The clinical importance of a case like the above is considerable, although at present there is not any special danger or even inconvenience to this patient. If in such a case a retropharyngeal abscess coexisted, or a suppurative peritonsillitis were to develop in the posterior position, the surgeon after having done his duty by giving vent to the

pus might easily find himself called upon to check a most embarrassing hemorrhage. It would be well for us to bear in mind that the condition in this child was only accidently discovered while she was being examined for an ordinary catarrhal inflammation.

Since the above report has been sent to the printers I have seen another case of apparently the same condition in a boy about seven years of age. The findings in this second case correspond so exactly the one just portrayed that another description would be superfluous. The enlarged artery was on the same side of the pharynx, though possibly somewhat more prominent.

THE TREATMENT OF PNEUMONIA.[1]

By Reynold Webb Wilcox, M.D., LL.D.,
Professor of Medicine and Therapeutics at the New York Post-Graduate Medical School and Hospital; Physician to St. Mark's Hospital.

The treatment of pneumonia always possesses great interest for the physician, both because of its prevalence and of its high mortality. Any method of treatment which can reduce the latter is well worthy of our consideration. What the mortality really is can be roughly stated as that about one-fourth of all patients attacked die. The laity regard this disease as even more fatal. Actual statistics vary much. Wells, in an analysis of nearly a quarter of a million cases, determined a mortality of a trifle over 18 per cent., the collective investigation of the British Medical Association 12 per cent., while of about forty thousand cases in the German army the fatality was less than 4 per cent. The significance of these figures lies chiefly in indicating the class of patients in which this disease is so fatal—the alcoholic subjects, debilitated and especially old individuals. I think almost any collection of mortuary reports will show that at least one-fourth of all necropsies will reveal chronic interstitial renal disease. Death more commonly takes place from gradual toxæmia or mechanical interference with circulation or respiration, the last being a gradual exclusion of air by the filling up of the ultimate air spaces of the lungs, and, as a consequence, compression of the capillaries. Another factor to be considered is the depressing effect of a great loss of blood serum if an extensive territory has been invaded by the disease. The dangers from the heart have always received attention at the hands of the clinician, and digitalis and other cardiac remedies enter into most schemes for the treat-

[1] Read before the Medical Society of the State of New York, at the Ninety-sixth Annual Meeting, 1902.

ment of pneumonia. For many years I have been impressed by the good effects obtained by the use of the nitrites in conjunction with strychnine, so ably advocated by Smith. The bleeding of the patient into his systemic arterial system by nitroglycerin or sodium nitrite, and the stimulation of the heart by strychnine nitrate, the loudness of the pulmonic second sound being taken as a guide, has been of remarkable service in my hands. Of late erythrol tetranitrate, in dose of one-half grain every four to six hours, gives more even and controllable effects than the evanescent nitroglycerin or the uncertain sodium nitrite. Strychnine I employ hypodermically as the nitrate, in doses from one-sixtieth to one-fifteenth of a grain every four to six hours, until I find the pulse satisfactory. Alcohol is only given to alcoholic subjects, and then with a free hand. Ammonium carbonate in ten-grain doses, in two ounces of milk every two hours, replaces strychnine in the aged. I believe that when we have employed these remedies with discretion and persistence we have done the most to meet the mechanical conditions that is at present possible. I am aware of the excellent results claimed for and obtained by veratrum viride in the very early stages of the disease occurring in young or even middle-aged robust individuals. However, the period of the disease and the class of suitable patients are far too limited in metropolitan practice to allow this to be considered as a proper routine treatment. I have seen brilliant successes and quite as often unfortunate results, and only exceptionally would I employ it at the present time. Bleeding is mentioned chiefly in the hereditary text-books, and to it the same remarks, perhaps more stringently, will apply. It is also a notorious fact that bleeding does not give as good results in pneumonia as in cyanosis with dilated right heart due to other causes. This observation leads to the questioning as to how much the pressure in the pulmonary artery is raised by extensive pulmonary consolidation. Clinically, the method above outlined has improved our results. Experimentally, Welch showed that it is very difficult to raise this blood-pressure unless extremely large areas of circulation were cut off. Quite recently Wood, Jr., has shown that various influences (among others, asphyxia) elevate the blood-pressure in the pulmonary artery, and this elevation is not a passive one. Experimentally, he shows that nitroglycerin and the other nitrites, while lowering the elevation of the aortic pressure, cause a slight elevation in the pulmonic system. Certainly at present the results of experiments do not very closely tally with those of observation.

In addition to the statement made above as to the unsatisfactory results of venesection, it should be stated that the effect of the toxins on the cardio-respiratory centres and of pyrexia on the heart-muscle must always be taken into consideration in discussing this difference,

and this brings us to the matter of toxæmia. There is certainly a considerable percentage of instances where the demonstrated area of consolidation during life and the evident amount of hepatized lung after death are absolutely too small for any mechanical theory to account for the fatal issue. Here toxæmia must be the efficient cause, and both prognosis and treatment should be largely influenced by our knowledge and appreciation of it. Before the modern concept of toxæmia had become established—in fact, before the microbic theory of pneumonia had been postulated—calomel was a favorite remedy. Later quinine, chloroform, and salicylic acid had a certain established reputation. In spite of all, the death-rate, if we may judge from hospital statistics, compared by decades, did not sensibly change. So late as 1897 Osler believed that we had no reliable measures at our disposal to combat the toxæmia of pneumonia. Within two years, however, Cassoute and Corgier reported that after continuous administration of fairly large doses of creosote carbonate (containing 91 per cent. of creosote, and made from it by the action of nascent carbon dioxide) in most cases a typical fall of temperature occurred during the first twenty-four hours of treatment, and if the remedy was persisted in for a sufficiently long period of time, the apyrexia became permanent. Relapses and sequelæ so frequently seen under other methods were entirely absent. So positive an assertion could not escape attention. Creosote —better beechwood creosote—is not a new remedy, but its caustic action and its irritating action upon the kidneys when given in necessary amounts had prevented its use. So pronounced were these untoward actions that I had abandoned its use in pulmonary tuberculosis several years earlier. The daily dose of creosote carbonate was from two to four drachms, the dose interval being six hours. So soon as the temperature reaches the normal the amount is reduced to one-half, and this is continued so long as auscultatory signs persist. What are the results? Cassoute and Corgier reported favorably upon eighteen cases; Stokes, seven; Bridges, eight; Frieser, nine; Meitner, thirteen; Eberson, four; Van Zandt, sixteen; Von Ruck, twenty (complicating pulmonary tuberculosis); Weber, nine; and Thomson eighteen cases. From these observations the statement of Van Zandt is fair—that creosote carbonate cuts short or aborts a large percentage, mitigates almost all the rest, and in a small percentage of pneumonia there is no result. Certainly if the early appearance of the crisis is any indication of the value of the treatment this remedy merits a careful trial.

My own experience covers thirty-three patients, with no deaths. The disease terminated by lysis in nine; by crisis in twenty-four. Crisis occurred on the sixth day in one, seventh in two, eighth in nine, ninth in six, tenth in three, eleventh in two, and on the twelfth day in one patient. In two patients above the age of seventy, lysis occurred.

Of three alcoholic subjects, in two lysis and in one crisis was noted. Two instances of double pneumonia, both terminated by lysis; in one the infection of the two lobes was contemporaneous, in the other by sequence. Aside from the remarkable reduction of mortality, the increased percentage of cases in which crisis is noted is suggestive as to the true significance of that phenomenon and is an argument for the value of the remedy in nullifying the bacterial activity and its results.

Of the accessory treatment, in addition to that with reference to the mechanical conditions, particular attention should be paid to the emunctories, in order that all avenues by which toxins can be eliminated, may be open. In patients suffering from renal disease of the interstitial variety, intestinal irrigations of a gallon of decinormal saline infusion, at a temperature of 108° F., practised twice daily through a rectal tube, is a most valuable method of provoking diuresis, stimulating the heart, and to a less extent, producing diaphoresis. Naturally, it thoroughly cleanses the large intestine. Intestinal antisepsis, as advocated by Waugh—one-sixth of a grain of calomel every hour for six doses, with saline laxatives enough to empty the bowels completely and keep them open afterward with from three to six grains of zinc sulphocarbolate every two to four hours, at other times than the calomel and salines, until the stools are odorless, and then just enough to keep them so—has been of frequent use and followed by marked benefit. It is true, however, that under the creosote treatment tympanites is rare, and the necessity for this remedy much lessened. Oxygen is of value if the respiratory surface is greatly decreased. It is perhaps at times used unnecessarily, but no harm results. In the earlier days of my practice cold applications to the surface were employed, but in recent years fever is held in less importance on the one hand, and cold water is now admitted to be a poor antipyretic, on the other, so that these have been abandoned. As for diet, milk, diluted with lime-water, or Vichy, or peptonized or fermented, should be our main reliance.

The present status of the treatment of pneumonia is especially satisfactory when results are considered. To summarize: 1. Continuous, persistent, and generous administration of creosote carbonate. 2. Careful adjustment of mechanical conditions. 3. Thorough evacuation of toxins by all possible ways. 4. Temporary supplemental oxygen by inhalation. 5. Liquid diet until physical signs disappear.

To be avoided, are antipyretics, opiates, ill-advised external applications and slowly-acting heart remedies, as digitalis.

A REVIEW OF THE STUDY AND TREATMENT OF HEATSTROKE AT THE PENNSYLVANIA HOSPITAL AND ELSEWHERE, 1751–1870.[1]

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Knowledge of the pathological effects of heat and of its literature has been acquired by most authors on these topics, in the reverse of the ideal order.

Unexpected experience has incited study that should have been preparatory. Such study has almost invariably resulted in the expression of two regrets—one, that so little was written; the other, that there was yet so much to know.

A liberal experience in 1892, the availability of case statistics dating from 1751, of meteorological data from 1831 and of case histories from 1873, at the Pennsylvania Hospital, all prompted not only their study, but also that of collateral publications. This reading betrayed that many authors wrote in ignorance of the work of their predecessors and of their contemporaries. Therefore the erroneous belief "that so little was written" justly gave way to regret that much which was written was so little known. This fact, and the completion by the Pennsylvania Hospital of its first century and a half of medical record, prompts the writing of this review and the publication, at a later date, of supplementary papers dealing either with other periods or branches of this topic.

The aim of this contribution has been to present for convenient reference, in chronological order, the observations, thoughts, and often the words of workers in this field during the period named.

INTRODUCTORY.

Biblical references[2] to fatality following exposure to the sun are followed after the lapse of centuries by the observations of Forestus,[3] in 1562, of Baglivi, in 1694, and of Lancisi, in 1705.[4] These three observers confounded the effects of heat with the symptoms of apoplexy, and this confusion was perpetuated by some observers up to 1852.

Forestus recommended, in the treatment of this condition, stimulating frictions, and observed that in many instances blood-letting was unne-

[1] Read in outline before the College of Physicians, June 1, 1901.
[2] J. J. Levick. The American Journal of the Medical Sciences, 1859, vol. xxxvii. p. 40.
[3] Loc. cit., p. 49.
[4] Loc. cit., p. 48.

cessary. He was the first to distinguish two types of cases, the mild and the fatal. Baglivi and Lancisi also recognized the predisposing influences of heat and humidity, and added that the depressing influences of prevalent war were contributory. On July 8, 1707, many people and animals were reported by Mosely[1] to have died in England from the heat of the sun. In 1738, in the early part of August, as reported by Dr. Lining,[2] the thermometer in the shade at Charleston, S. C., rose successively for some days to 98° F., and "several persons died of apoplexies." In 1743, from July 14th to 25th, 1100 people are reported by this authority to have perished in the streets of Pekin from the same cause.

Periods I. and II.—1751–1810.

Diagnoses of Apoplexy, Effects of Drinking Cold Water, and Coup de Soleil Confounded.

In 1769, eighteen years after the founding of the Pennsylvania Hospital, its records show the first entry of the pathological effects of atmospheric heat—under the diagnosis of "hurt by drinking cold water." This case was that of a woman who spent two days in the hospital. Fifteen similar entries with such variations in phrasing as "convulsions" or "inflammation of stomach from drinking cold water," making a total of 16 cases and of 3 deaths, the first of which is recorded on the 31st day of the 8th month, 1793 (see Graphic and Statistical Chart in later publication), are entered on the hospital register prior to 1831, when the phrases "Effects of Heat" (May 1, 1831) and *coup de soleil* (June 1, 1831) appear for the first time, showing an advance in the etiological appreciation of this condition. The last entry of "drinking cold water" is made on June 10, 1838.

The diagnoses having in common a reference to "cold water" were, doubtless, made or directed by subscribers to the popularly accepted teaching on the subject by Dr. Benjamin Rush, who, however, was not associated with the hospital as one of its visiting physicians at the time of the first cold-water diagnosis noted—1769—but became so, fourteen years later, on May 26, 1773, and so continued until his death April 19, 1813.

In 1789 Dr. Benjamin Rush[3] published in London his *Medical Inquiries and Observations*, one of which is entitled *An Account of the Disorder Occasioned by Drinking Cold Water in Warm Weather, and the Method of Curing It.*

It had been noted by Dr. Rush that in some summers four or five

[1] Mosely. Tropical Diseases, London, 1803, p. 57.

[2] Dickson. Observations on the Effects of Heat. The American Journal of the Medical Sciences, 1828, vol. iii. p. 268.

[3] Rush. Medical Inquiries and Observations, 2d edition, Philadelphia, 1789, p. 150.

persons, usually laborers, died suddenly in one day subsequent to drinking pump water. The following paragraphs present the pith of his observations:

"Three circumstances generally concur to produce disease or death from drinking cold water: 1. The patient is extremely warm. 2. The water is extremely cold. 3. A large quantity of it is suddenly taken into the body." The danger is in proportion to the degree of combination of the three circumstances cited.

"In a few minutes after the patient has swallowed the water he is affected by a dimness of sight; he staggers in attempting to walk, and unless supported, falls to the ground; his nostrils and cheeks expand and contract in every respiration; his face appears suffused with blood, and of a livid color; his extremities become cold, and his pulse imperceptible; and unless relief is speedily obtained the disorder terminates in death in four or five minutes.

"*I know* of but one certain remedy for this disease, and that is *liquid laudanum*. The doses of it, as in other cases of spasm, should be proportioned to the violence of the disease. From a teaspoonful to near a tablespoonful have been given in some instances before relief has been obtained."

Patients were directed—

"1. To grasp the vessel out of which they are about to drink for a minute or longer with both hands; that a portion of heat may be abstracted from the body and imparted to the cold liquid.

"2. If they are not furnished with a vessel to drink out of, but obliged to drink at a pump or spring, always to wash their hands and face, previous to drinking, with a little of the cold water. By receiving the shock of the water first on those parts of the body, a portion of its heat is conveyed away, and the vital parts are defended from the action of cold."

The theory presented and the treatment advised by Dr. Rush in relation to the observations quoted was subscribed to by partisans and controverted by opponents, who also contended much among themselves, and who—with their posterity—may be roughly classed as follows:

I. Those who, like Dr. Rush, failed to distinguish the effects of heat and those of all kinds of *immoderate drinking*.

This class may be thus subdivided:

1. Those who agreed with Dr. Rush's theory and treatment of the effects of cold drinks upon overheated and exhausted persons.

2. Those who differed with Dr. Rush upon these topics.

II. Those who contended that the "disorder" described by Dr. Rush was due to *the effects of heat*, of which thirst and drinking is a symptom and not a cause.

This class contains vitally differing beliefs:

1. Those who held that heat effects were always *congestive*, that they frequently resulted in apoplexy and that the condition must always be met by depletion. This is the earliest teaching in this matter, and it had its supporters as late as 1856.

2. Those who deemed heat effects to invariably result in *exhaustion* demanding routine application of heat, administration of stimulants, and without exception contraindicating blood-letting.

3. Those who saw that heat effect and apoplexy were diseases apart, and also that heat caused at least two widely differing conditions:

a. Febrile or congestive, demanding the application of cold and sometimes benefited by depletion.

b. Exhaustion—indicating rest, sometimes stimulants and artificial heat, and always contraindicating blood-letting.

This classification represents the principal differences of opinion that have obtained at various periods down to the present day; it necessarily fails to take account of several composite beliefs, and contains many subdivisions. Each subdivision of Class II. may again be subdivided, according as its exponents thought that direct *solar* heat, or knew that its indirect form and that *artificial* as well as solar heat, would produce the results observed.

In 1798 Dr. James Currie[1] published in his *Medical Reports on the Effects of Water* a chapter entitled "On the Disease that Arises from Drinking Cold Liquid, or Using the Cold Bath after Severe Exercising." Like Dr. Rush, he failed to note that the accompanying atmospheric heat, in the majority of the cases cited, was the exciting and not, as they supposed, merely a predisposing cause. He otherwise elaborately differed from Dr. Rush. According to Dr. Currie's theory of the fatal phenomena reported, the latter occurred not because the individuals drinking were overheated, but because they had been overheated. They had, he supposed, paused after their exhausting exertions, had begun to chill by perspiration, were in a condition of depression fatally increased by the draught of cold water—that could have been taken with impunity in the heat of exercise. He, therefore, considers the precautionary suggestions of Rush—because of their nature chilling—as contributory to and not preventive of evil. That this scholarly physician in Liverpool should at that period fail, in reading, to detect symptoms incident to torrid temperature outside of his experience, is hardly surprising. There were, however, those who, with advantages of observation, correctly divined heat to be the cause of the supposed cold water malady, and classified its manifestations.

[1] Medical Reports on the Effects of Water, Cold and Warm, as a Remedy in Fever and Other Diseases, Whether Applied to the Surface of the Body or Used Internally, Including an Inquiry into the Circumstances that Render Cold Drink or the Cold Bath Dangerous to Health, etc. James Currie, M.D., F.R.S., Liverpool, 1798, p. 95.

Period III.—1811–1841.

Supposed Cold Water Effects Recognized as Coup de Soleil, Which Is Still Confounded with Apoplexy.

In 1811 Hillary's[1] work on *Tropical Diseases* appeared, with notes by Benjamin Rush. It states that upon the change of the weather from very dry and warm to very moist and warm "dysenteries became very frequent;" "and some were seized with apoplexies and palsies, and some of the first died before any proper assistance could be called in." In a foot-note he queries: "Were not the apoplexies and palsies also produced by the same causes, viz., a diminution or stoppage of the perspiration?" etc. This report is replete with meteorological data; but some of the cases referred to evidently resulted from heat and humidity, and not from apoplexy.

In 1814 Dr. Benjamin H. Coates "inhabited the Pennsylvania Hospital," as he reported in 1858, in his discussion[2] of Dr. Levick's "Remarks on Exhaustion from Heat (Sunstroke)," before the College of Physicians. He recalled that at this time, by order of the Humane Society, the public pumps were posted with cautions against sudden death from drinking cold water. He saw many persons at the hospital (Pennsylvania) alleged to be suffering from a spasmodic affection of the stomach, described by Dr. Rush as a result of drinking cold water; but it was Dr. Coates' opinion, and that of some medical attendants at the hospital, that these patients were usually under the influence of ardent spirits and suffered rather from congestion of the brain than that of the stomach.

Speaking of the cases in 1814, Dr. Coates goes on to say: "The cephalic cases received little other treatment than rest, silence, a gloomy room, *cold to the head*, mild stimulants to remote parts of the skin, laxatives, and a cool abstemious and sedative diet." This is the earliest mention found of the use of cold in these cases. He comments that the great terror of the destructive effects of the use of ice in drinks, etc., and its apparent dangers were then (1858) forgotten. He had not examined the hospital records, but had no recollection of any death in such cases at that period, 1814. The hospital records are confirmatory (see chart in later statistical publication). The cases could not have been of grave character.

In the winter of 1818, Dr. D. Francis Condie, as he reported in 1850 to the College of Physicians, in his discussion of Dr. Pepper's communication on "Coup de Soleil," attended a "colored preacher" seized in the

[1] William Hillary. Observations on the Changes of the Air and the Concomitant Epidemical Diseases in the Island of Barbadoes, etc., 2d edition, London, 1776, p. 18, with notes by Benjamin Rush, Philadelphia, 1811.

[2] Transactions of the College of Physicians of Philadelphia, 1858, n. s., vol. iii. p. 235.

midst of his exhortation, in a crowded and overheated church, by convulsions alternating with tremors. Dilated pupils, thready pulse, and clammy surface were accompanying conditions. He says: "In this case the disease was produced by the exhausting effects of undue exertion in a heated and confined atmosphere; and he believed that in those who are attacked while laboring in the open air, during the intense heat of summer, it is also produced by exhaustion, and not by any direct influence of the sun's rays, as some have supposed." Dr. Condie's narration of this case and his comments upon it place him not only as the first to distinguish between congestion and exhaustion from heat, but also as the first to recognize that heat alone was the essential cause of the phenomena the which for many years later were attributed to the sun alone. In this regard it is interesting to refer to Dowler's explicit and contrary statement in 1841 (p. 496 of this review), also that Dr. Geddes[1] ("Clinical Observations on Diseases of India, 1846") relates a case of heat effect occurring in a convalescent from measles, who was in hospital and not exposed to the sun.

In 1818 Dr. John Watts, Jr.,[2] submitted to the Medical Society of the County of New York, "Remarks on the Supposed Effects of Drinking Cold Water; Illustrated by Cases which Occurred During the Hot Weather of the Summer of 1818."

This paper is of peculiar interest, as the first published protest against attributing to water the effects of heat. The reminiscences of Dr. Coates referring to 1814 were not recorded until 1858. The theory voiced by Rush was popularly accepted. This initiatory attack, though not without fault, was well prepared and pushed far, but it took many others to eradicate an error which was last defended in 1859.

The author most ably and fairly reviews and discusses the cold water controversy, quoting Rush,[3] Currie,[4] and Jackson.[5] He takes ground: that the "usual effects" of intemperate water drinking are not denied, nor altogether its agency in producing the symptoms noted; that an "ardent sun" independent of water drinking is in itself sufficient to cause "*coup de soleil*, or sunstroke;" that the symptoms of the latter are "analogous" to those attributed that summer to the effects of cold water; that the contention of the author had the support of reputable physicians who characterized patients suffering from this disease as "rum and sun cases;" that although the disorder described by Rush terminates by "death in four or five minutes," yet no "sudden death" had been heard of from this cause; that laudanum was not, in this regard, a specific remedy, since no recovery was attributed to its sole influence, whereas, many cures were

[1] Wood. Sunstroke, Philadelphia, 1872, p. 10.
[2] John Watts, Jr. The Medical and Surgical Register, New York, 1818, p. 81.
[3] Rush. Loc. cit. [4] Currie. Loc. cit. [5] Jackson on Cold Water, p. 377.

noted in sequence to copious bleeding; finally, that there was reason to fear that some persons fell "victims to the injudicious and extravagant administration of laudanum." The abuse of opium at this time in the treatment of heat or cold water effects was probably the cause of this helpful remedy falling into disfavor until reintroduced in 1868 by Drs. Hutchinson and Norris (p. 520 of this review).

The symptoms he cited of the group of 13 cases reported are typical of the major form of heat effect. It is of interest to note that: "The heat of the body was likewise remarkably great, and in some instances continued for a long time after bleeding."

Two of the 13 cases reported, died; but one was examined by autopsy, and it exhibited "about five ounces of extravasated blood on the surface of the dura mater." This case, apparently of true apoplexy, naturally confirmed Dr. Watts in his prejudice that the pathological result of heat effect was apoplexy.

The scheme of treatment included: "laudanum, æther, and other stimulants," "when the apoplectic symptoms had not yet supervened," the influence of these agents was but "momentary," apoplectic symptoms developing in a short time; the rubbing of aqua ammonia over the whole body, a measure credited with lessening the bodily heat as well as arousing the patient from an apparently fatal torpor; and, finally, large and repeated bleedings. It was upon blood-letting that Watt placed most reliance. Several of his patients were bled of sixty, and one of eighty ounces in two hours.

As he inveighed against overdosage with laudanum, so did those following him unsparingly condemn routine and reckless blood-letting.

In 1821 the British frigate "Liverpool,"[1] when in the Persian Gulf, lost "from a species of *Coup de Soleil*," three lieutenants and thirty men in one day, although double awnings were stretched and the decks kept wet.

In 1824, as reported by Dr. Samuel H. Dickson[2] in 1828, nine consecutive days on which the temperature was 90° F. or above, preceded by six days in which the temperature was 90° F. or near it, resulted in 34 deaths from "apoplexy and insolation" in Charleston, S. C.

In 1826 Dr. Daniel J. Carroll,[3] presented his "Observations on the Morbid Effects Produced by Drinking Cold Water." This article, in spite of the contrary desire announced in the introduction, admirably presents the confusion of ideas prevalent upon the effects of heat at this time. It begins as follows:

"Notwithstanding the number who are supposed annually to perish from this cause (referring to the title), yet there are many cases which

[1] Wellsted's Travels to the City of Caliphs.
[2] Dickson. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1828, vol. iii. p. 271.
[3] Carroll. Philadelphia Journal of the Medical and Physical Sciences, 1826, vol. xiii. p. 122.

result from heat alone, or exposure to the direct rays of the sun, producing the *coup de soleil* of the French. Other forms of cerebral affection, from simple congestion of the brain to apoplexy (though as discordant with this, in their nature as mode of cure), have, nevertheless, been erroneously attributed to cold water," etc.

In spite of the avowed intention of the paper to distinguish heat from cold water effects, it details with evidences of much unconsciousness and erudition undoubted cases of sunstroke as due to cold water drinking. An interesting feature of the symptomatology is the mention of a vesicular eruption. Like Watt, he comments upon much fatality resulting in mild cases from overdosing with laudanum."

In 1828 Dr. Samuel H. Dickson[1] varied the monotony of title favored by foregoing authors on this topic by publishing the first paper avowedly dealing with "Observations on the Effects of Heat." Reference to the experimental endurance of extraordinary temperature without evil effect is followed, first, by a study of the phenomena and treatment of the condition now popularly known as spring fever, and second, by a report of the cases of insolation and *coup de soleil* from the persistently hot weather in 1824 already noted. His tribute to the cold water theorists is to advise the drinking of cold water in hot weather.

Without having witnessed post-mortem section of a case of heat effect, he concludes the condition to be one of venous engorgement tending to apoplexy. He divides the cases he reports into two classes, the apoplectic or cerebral, and the phrenetic or meningeal. He cites in confutation of Rush a case of apoplexy supposed to be produced by heat, a mulatto with subsequent hemiplegia, who immediately preceding his attack drank water the temperature of which Dr. Dickson found to be about the same as that of the patient. The inference being —that thirst is a symptom marking the onset of the attack. As the water was not cold, it could not, as supposed by the bystanders, have excited an attack of the disorder described by Rush. The coincidence of this case occurring on a warm day in a man laboring in the sun was apparently confirmatory of the theory that heat is productive of apoplexy. On the other hand, cerebral hemorrhage has not been observed at autopsy in cases of heat effect, and the latter most rarely results in hemiplegia.

The cases described as apoplectic were bled, and those classed as meningitic were treated by cold affusions poured from a height on the head of the patient, who was raised to a sitting posture. The return of symptoms calling for a repetition of the affusion is the first record found of what still proves a valuable aid in treatment. The author saw "in

[1] Dickson. Observations on the Effects of Heat. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1828, vol. iii. p. 268.

one day five persons, three of whom recovered entirely under this simple means of cure." If the recovery was not thus complete, and mania and phrenitis supervened, the cases were for the most part manageable, but now a free use of the lancet became necessary, and purgatives of the *most active* power were demanded.

In 1830 Dr. William W. Gerhard,[1] later a visiting physician to the hospital, was the first to record negative post-mortem appearances of the brain in cases that had died from the effects of heat. His observations are thus presented:

"Symptoms of the same character occur from the effects of heat upon the nervous system during the warm season. During the intensely hot weather of the summer of 1830 I witnessed the opening of the bodies of twenty or thirty persons who died from this cause. We found no organic disease of the brain, but merely a slight congestion—such as is observed in other acute diseases—*which it would be idle to set down as a cause of death.* These were the appearances in those only who died suddenly of exposure to heat; for if time elapses for reaction to come on, inflammation of the brain may take place, but it is then a secondary affection."

In 1830 *The Boston Medical and Surgical Journal,*[2] No. 17, June 8th, reprints the paper of Dr. Rush already quoted (p. 486), and in No. 18, on June 15th—the next issue—publishes a protest to the theory of Rush by Dr. F. J. Higginson, who supports the views of Currie,

In 1831 meteorological observations were begun at the Pennsylvania Hospital, and they continue to the present day. The Government Weather Bureau was not established until many years after that of the hospital. The relation of atmospheric temperature to the occurrence of illness from heat will be considered in a later and statistical publication on this topic. It is of interest to note that the institution of atmospheric observations at the hospital, in 1831, is coincident with its first recorded diagnosis of "Effects of Heat" and *coup de soleil,* such cases prior to that time being attributed to the effects of "drinking cold water."

In 1831 Andral,[3] of Paris, under the classification of "The Eighth Form of Cerebral Congestion," notes a definite thermometric relation between high atmospheric temperatures and the occurrence of symptoms that he classes with cerebral congestion and apoplexy. He reports a number of cases, without fatality, occurring in young soldiers and treated at La Pitié. He also described the post-mortem conditions in

[1] Graves' System of Clinical Medicine, with Additional Lectures by William Gerhard, Philadelphia, 1848, p. 677.

[2] Boston Medical and Surgical Journal, 1830, vol. iii. p. 265.

[3] G. Andral. Medical Clinic, or Reports of Medical Cases. Translated by D. Spillan, Philadelphia, 1838, p. 108.

two of three undoubted sunstroke cases, and noted in both of them petechiæ, liquid blood, pulmonary, cerebral, and venous engorgement, and in one of them that had *contracted heart*, which later was observed experimentally by Bernard, in 1859, and by Wood at autopsy, in 1863. No cerebral or meningitic effusions were discovered.

In 1836 the first English report of congestion of the lung as a post-mortem observation, in 1834, in the cases of three soldiers, was made by J. J. Russell,[1] of the Seventy-third Regiment, English Army. He was surprised to find no congestion of the brain. It must be remembered that even up to 1856 the effects of heat were confounded with apoplexy.

The exciting cause was exposure to the sun at the funeral of a general officer at Madras, India; the predisposing causes were red coats and military stocks. Some eighteen men were overcome. Three died promptly where they fell, and two others some hours later. In the fatal cases the symptoms and post-mortem appearances were alike. The symptoms reported were "*excessive thirst*, and a sense of faintness; difficulty of breathing, stertor, coma, lividity of face," and one examination of the pupils of one man, found them contracted. The autopsy discovered neither congestion nor accumulation of blood in the brain. The lungs were congested to blackness, there was a collection in the right heart and the adjoining vessels. "The chief seat of mischief was in the chest and not in the head."

In 1836 Dr. Robert M. Huston,[2] as stated in his discussion of Dr. Pepper's paper of 1850, witnessed four post-mortem examinations of men sunstruck while working in a culvert, and was amazed to find no congestion of the brain.

In the period of 1811–1814, fifteen cases of heat effect were treated at the hospital, with a mortality of three. The recognition by Condie, in 1818, that artificial heat and the indirect as well as the direct rays of the sun were exciting causes of what was then known as *coup de soleil*, or heat apoplexy; his perception that the effects of this condition were chiefly twofold and widely differing in form and treatment; the discovery by Gerhard, in 1830, that, contrary to centuries of conviction, the pathology of the condition was quite distinct from that of apoplexy; the report by Andral, in 1831, of petechiæ, of liquid blood, of pulmonary, cerebral, and venous engorgement, and of contracted heart as the characteristic pathology of this condition, which he did not interpret correctly, but which was later verified as the true morbidity of this state by Russell (1834), Bernard (1858), H. C. Wood (1863), and Stiles (1864)—these are the most noteworthy discoveries of this period.

[1] J. J. Russell. The London Medical Gazette, vol. xviii. p. 71.
[2] Transactions of the College of Physicians, 1850.

Period IV.—1841–1870.

Heat, Artificial as Well as Solar, Experimentally Demonstrated the Exciting Cause. Success of Refrigerating Treatment.

In 1841 Dr. Thomas M. Markoe[1] reported among a number of cases treated at the New York Hospital, one, Case IV., entitled "Congestive Apoplexy from Drinking Cold Water," the history and post-mortem examination of which classify it as a fatality from heat. The first use of ice water affusions is recorded in the treatment of this case. Autopsy, eighteen hours after death, discovered severe venous congestion of the brain, the sinuses to be swelled with darkened blood, the membranes to be darkened, and the brain on section to exhibit bloody points. There was no extravasation. Blood flowed from the divided vessels at the base of the brain. The complete details of this case help to accentuate one's surprise at the diagnosis.

In 1841 Dr. Bennett Dowler,[2] of New Orleans, published a paper, entitled "Solar Asphyxia, Coup de Soleil, or Sunstroke." It begins as follows: "In the numerous works written or republished in the United States, I have not seen any satisfactory notice of this very frequent and, in the South, most fatal of all maladies." Ten and nineteen years later Drs. Pepper and Levick, respectively, regret the lack of literature on this topic even at those later dates. Dr. Dowler's paper is remarkable for its breadth and detail of observation. As the isolated study of one man it is very accurate and long antedates corroborative publication by others. Two of its last pages (215, 216) are well devoted to a clear differentiation between sunstroke and apoplexy, which for years later were confounded commonly. The name *Solar Asphyxia* was adopted by him "several years before" he "had an opportunity of testing by dissections the character of the disease, which"—he claims—"it so fully expresses." He adds, "*Pulmonary apoplexy* is a name more characteristic of the morbid appearances of the lungs, but it makes no allusion to its solar origin or to the symptoms of suffocation which mark its progress."

Thus, knowledge of the congested condition of the lung in the major form of heat effect was recognized clinically by Dr. Dowler before its verification by him at autopsy. His observations were more detailed and comprehensive than those of Dr. Russell, made in 1834 and published in 1836. Dr. Dowler states his observations and nomenclature to antedate, by several years, their publication in 1841. The earliest case he cites, he attended in 1836.

[1] T. M. Markoe. A Report of Cases Treated in the Second Surgical Department of the New York Hospital. The American Journal of the Medical Sciences, 1841, n. s., vol. ii. p. 343.

[2] New York Medical Gazette, 1841, vol. i. p. 209.

He next presents the practical importance of the classification of solar diseases, and submits the following: 1. Solar exhaustion or syncope. 2. Solar or sun pain. 3. Solar excitement or inflammation. 4. Solar asphyxia, which he makes "the principal subject of investigation." He first distinguished between the two extremes, saying: "Solar exhaustion differs from solar asphyxia, both in symptoms and treatment. In solar asphyxia the skin is extremely hot, and generally dry; there is a choking sensation and a total loss of sense. In solar exhaustion, the skin is moist, pale, and cool; the breathing is easy, though hurried; the pulse is small and soft; the vital forces fall into a temporary collapse, the senses remaining entire." The conditions described are, undoubtedly, those we know to-day respectively as *heat* or *thermic fever*, and as *heat exhaustion*, and the treatment advised for solar exhaustion is the same as that now in use for heat exhaustion. This is the first publication of these distinctions of the results of heat. Dr. Condie assigns his *recognition* of them to 1818, but he did not *publish* them until 1850, and he correctly ascribed the conditions observed to heat, whether of solar influence or not. Whereas, Dr. Dowler says: "Solar and terrestrial heat differ essentially in their action; on health the latter seems never to produce any morbid effects, resembling sunstroke, though many firemen in boats, foundries and furnaces are exposed to a high temperature." On the other hand, Dr. Condie lacked Dowler's clear conception of the differences between heat effect and apoplexy. This is apparent in Dr. Condie's discussion of Dr. Pepper's paper, when he says: "That the direct influence of intense solar heat may produce congestion of the brain and apoplexy, Dr. Pepper admits, and Dr. Condie has known it to produce well-marked meningitis."

To return to Dr. Dowler's classification of solar diseases, he thus defines Classes II. and III.: "II. Solar, or, as it is commonly called by the people, *sun pain*, is a chronic disease in which the abdominal organs are more or less deranged, though the most remarkable symptom is a pain in the head while the sun is above the horizon." "III. *Solar excitement* or *inflammatory reaction*, sometimes follows solar asphyxia of the second degree, being attended with febrile heats, arterial throbbings and headaches." Classes II. and III. would be considered, at the present day, as different degrees of meningeal congestion or inflammation from heat.

Want of acclimation he considers strongly predisposing to solar disease, which he pronounces an urban disease affecting those not thoroughly acclimated to the city. The most thoughtful explanation of why heat effects are chiefly confined to large cities is furnished by Dr. Lente (p. 501). Dr. Dowler quotes the great fatality on the "Liverpool" (p. 491) as an instance of lack of acclimation. He notes that he has seen but one negro die of sun exposure—that the disease is practi-

cally confined to males; females, comparatively, being unexposed to the sun. He knew only of one woman to be affected, she happened to be a negress, and recovered. But even the negro, though not prone to fatality, required acclimation. "Slaves from Missouri, Virginia, and Maryland suffer as much if not more from debility than the whites during the first and second summers." "Even horses are debilitated, often get the *thumps*, and frequently die from solar asphyxia during the acclimation period. This is so well known that an acclimated horse or mule is worth much more than one not so protected."

Among his enumeration of the predisposing causes of solar asphyxia —*i. e.*, heat or thermic fever—are a full meal, unsuitable clothing, and fatiguing labor. He erroneously considered exposure to the direct rays of the sun as the only exciting cause of the symptoms he witnessed. That the same phenomena could result from artificial heat was outside his experience. He explains that the man struck down in the street with solar asphyxia is commonly mistaken for an apoplectic, some of the bystanders put ice to his head, "and the first bleeder that can be had performs blood-letting." He records a case of solar asphyxia that he bled with successful result at the instant of onset, also a case of solar excitement—meningitis—that it cured. Referring to the first case, he says: "In this case, blood-letting was probably useful, but in nine cases out of ten it is useless, if not worse, accelerating the death from five to fifteen minutes. During the last five years I have been called to see a very considerable number of sunstruck persons, within five or ten minutes after they fell in the streets; formerly I used to bleed them, and, though *the great heat of the body is thereby suddenly diminished*,[1] the pulse becoming soft as air, yet by the time the arm is tied up (which is done more for form than necessity), the patient is choked suddenly, and to appearance, by a tenacious mucus, the breathing not ceasing gradually as in other disease, but instantly, the face turning livid, and even its veins, especially upon the forehead, becoming at the moment distended. Bleeding hastens the strangulation, though it is always desired by the friends."

While he condemns bleeding in general he states that its instant performance is the only possible hope of those stricken with solar asphyxia of the first degree, which he considers as practically fatal and within half an hour if the onset occurs in the most heated part of the day. The application of ice to the head and the pouring of ice-water over the head and neck are enumerated in conjunction with general sinapisms as methods of treatment employed in solar asphyxia, but they are not specifically commended. This is the first mention I find of the use of ice and of ice-water affusions. He gives an elaborate

[1] Italics introduced.

description of the symptomatology of solar asphyxia and notes as do other early observers the inability of the grave cases to swallow. He expresses the opinion that it was hurtful to attempt medication by mouth as increasing the tendency to strangulation. "In the act of dying, he noticed a slight curving of the body laterally,[1] with a feeble contraction of the fingers. In solar asphyxia, the symptoms and the manner of death are more uniform than in any other malady. *The heat of the body, both before and after death, is a most remarkable circumstance.*[2] In the hurry incidental to a death so sudden I have not had an opportunity of applying the thermometer, to ascertain the exact temperature; but judging from the sense of touch alone it would seem greater than in the hottest fevers. The heat may be felt, radiating from the patient's body, at a distance of two or three feet. In cases where the patient has not been bled copiously the heat is very pungent. The heat of the body continued, generally many hours after death, including the whole night. This is the more remarkable, as our nights are not hot and sultry, but accompanied with breezes, which, by morning, cool even the walls and pavements."

"*After the death of the lungs, or the cessation of respiration, the heart and arteries will, in some instances, continue to act.*"[3]

This remarkable paper, which also contained much data here unnoticed, and the report of several cases, the first occurring in 1836, thus concludes: "The solar heat probably accumulates upon the surface of the body faster than nature can refrigerate through the lungs and the skin by evaporation; inequilibrium presses upon the vital energy, which, being exhausted in the contest, as well as by excessive labor, is unable longer to neutralize the excess of temperature—often 40° F. more than that of the body. Vital chemistry is unequal to the task of preventing the conduction of heat into the body, and death is the consequence."

The explanation of "the vital chemistry" was not successfully attempted until it was begun by Claude Bernard, in 1858, carried further by Stiles in 1864, and far advanced toward completion by Dr. H. C. Wood in 1872 (see pp. 509 and 514).

In 1845 Dr. Dowler,[4] who had for some time been studying the antemortem and post-mortem thermometry of yellow fever and of many other diseases, published in September the following most interesting observation, both medically and historically:

On July 24, 1845, he observed in a case of "solar asphyxia," twenty minutes before death, an axillary temperature of 111° F. One hour

[1] Lateral convulsive contraction was noted by the reviewer in one case observed by him in 1892.

[2] Italics introduced.

[3] This opportunity occurred in 1845, and the temperature then recorded was the first reported of cases suffering from heat effect.

[4] The Medical Examiner, Philadelphia, 1845, n. s., vol. i. p. 526.

after death the thermometer registered 113° F. From that time the cadaveric temperature began to decline. Nine hours after death the temperature was 109¾° F. The observer makes the following comment:

"I have had much experience in this malady, but never happened to take my thermometer with me before; but I am convinced that solar asphyxia, in the first degree is the hottest of all maladies, and can hardly be less than 120° F. in many cases! The patient seldom has more than *one premonitory symptom—a sudden dryness and heat of the skin.* I suppose that this man's heat was below the average; certainly not so great as some others, according to my touch."

This seems, without doubt—the language is so explicit—the first thermometric observation of heat effect made by Dr. Dowler, although Dr. Levick[1] credits Dr. Dowler with the record of an ante-mortem observation of 99° F. and a post-mortem observation of 104° F. in a case of heat effect on May 3, 1844, a year earlier than the case cited, but no reference is given. Either is as far as the reviewer knows, the first thermometric observations ever made of the effect of atmospheric heat on man.

In 1846 Dr. Geddes[2] reports a case of heat effect developing in a convalescent from measles in a hospital ward. This is the first published report of heat effect not caused by solar heat. The case reported by Condie is of prior date, 1818, but was not published until 1850.

In 1846 Dr. Israel Moses[3] published a report entitled "Exhaustion by Heat and the Effects of Cold Drinks when Heated"—the context suggests *when the drinker is heated*—was meant. Sixteen cases are reported. They are classified, in brief, as cases of apoplexy, exhaustion, and effects of drinking cold water.

As in the paper of Dr. Carroll (p. 491), the prefatory classification is not carried out, and detailed histories show that undoubted cases of heat are erroneously considered as cold-water effects, Rush and Currie being quoted. Six of the cases died, of which four were bled. The author considers bleeding contributory to fatality, and advocates treatment by the hot bath 104° F. to 108° F., "aloetic enemata," sinapisms and frictions.

The six fatal cases were examined post-mortem, the lungs being congested in five, and not markedly so in Case X. The brain was found congested in Cases I. and IV., the membranes in Case VI., Cases VII. and VIII. were too much decomposed for examination; in Case X. no marked congestion was observed.

[1] Pennsylvania Hospital Reports, 1868, vol. i. p. 382.

[2] Geddes. Clinical Observations of Diseases of India. London, 1846. See Sunstroke, H. C. Wood, Philadelphia, 1872, p. 10.

[3] Israel Moses. The New York Medical and Surgical Reporter, 1846, vol. i. p. 11.

In 1849 Dr. Henry Hartshorne was the first of the staff of the Pennsylvania Hospital (he being a resident physician) to publish a report[1] of *coup de soleil*. He described four cases treated in 1846; two were fatal, the second was a chronic alcoholic, and the third died on admission. He notes that several other cases occurring in the city at the same time all proved fatal. His treatment is advanced and interesting, and he records the first use at this hospital of ice and of the cold douche to the head. Two of his cases were bled, but with difficulty, by cupping at the back of the neck: one died, the other recovered. Sinapisms were applied to the abdomen and legs. Aromatic spirits of ammonia were given by the mouth, and turpentine by the bowel. One case, while having cold applied to the head, was placed in a warm sitting bath.

In 1850[2] Dr. William Pepper, the elder, reported before this College twenty cases treated at the Pennsylvania Hospital in the seven[3] preceding years. Of these ten died, seven recovered, and three, partially recovered, became insane. All of these cases had been bled before admission to the hospital. Dr. Pepper reports symptoms and post-mortem appearances. Both these conditions were possibly modified by the bleeding to which the patients were subjected before being admitted into the hospital. The "pallid, flaccid, and softened" condition of the heart noted in four cases by Dr. Pepper and reported in one by Dr. Reed in discussion, were also due, probably, as suggested by Dr. Stiles,[4] to post-mortem change, the autopsies being made six to eight hours after death. Autopsy was found impracticable by Dr. Levick[5] ten hours after death. Dr. Horatio C. Wood[6] makes the following suggestion in regard to these cases. "The cases were very probably true sunstroke and not simply heat exhaustion, as I have suggested, and the cool skin was the result of the bleeding. The soft, flaccid hearts were found because the temperature not being high at the time of death the cardiac myosin did not coagulate."

Dr. Pepper says: "The disease is evidently one of nervous exhaustion. The cases of apoplexy and congestion of the brain occasionally produced by insolation should be carefully distinguished from the cases above alluded to, the pathology and treatment of the two being very different." Again: "The cases we usually meet with during the intense heat of our summers present no symptoms similar to those which result from cerebral congestion, and demand for their cure stimulants

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1849, n. s., vol. xviii. p. 66.
[2] Ibid., 1851, n. s., vol. xxi. p. 183.
[3] The hospital records for this period, and for ten preceding years, show the admission of but fifteen cases, six of which died and nine recovered.
[4] Boston Medical and Surgical Journal, 1864, vol. lxx. p. 349.
[5] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1859, vol. xxxvii. p. 49.
[6] Wood. Sunstroke. Philadelphia, 1872, p. 107.

instead of bleeding." Dr. Pepper closes by commenting on the absence of satisfactory medical literature on the subject at that day. This was the first presentation of this topic to the College of Physicians, and it is also the first, though not a very clear report by a member of the staff of the Pennsylvania Hospital and by a Fellow of the College of the difference between exhaustion and congestion following exposure to the sun.

Dr. Condie, in discussion of Dr. Pepper's remarks, gives a most clear and differential description of the symptoms of exhaustion from heat, and also—which Dr. Pepper did not—of congestion (fever) from heat. As already cited (p. 489), his recognition of these conditions dates back to 1818, and has such priority but not that of publication. He also makes distinction in the treatment, condemns bleeding for heat exhaustion, and, on the other hand, tells how he saved life by bleeding in a case of heat congestion accompanied by convulsions. His belief that heat effect may result in apoplexy has already been noted (p. 496). Post-mortem examination of the brain convinced him that "these cases are dependent upon acute meningitis." His is also the first recognition that artificial heat as well as solar heat can produce the condition at that time only attributed to the sun.

Dr. Reed also took part in the discussion, reporting a case which was subsequently examined post-mortem, a foot-note states that the heart in this case was flaccid.

Dr. Parrish closed the discussion. While acknowledging the value of Dr. Pepper's observations, he noted that they were in opposition to the prevalent doctrines of congestion and inflammation applied to all violent diseases which destroy life.

On page 536 of the same volume of THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, Dr. Frederick D. Lente, resident surgeon at the New York Hospital, answers Dr. Pepper's expressions of surprise that there was so little and such vague literature upon this subject: The victims of heat are principally laborers in cities "who earn their daily bread by their daily work, and are consequently compelled usually to labor during the intensest heat of the day, when the thermometer in the shade ranged from 96° F. to 100° F., and over, with not only the direct rays of the sun playing upon them, but also the reflected rays from the pavements and buildings. Added to this as the efficient and exciting cause, we have fatigue, intemperance, and often insufficient or improper food as predisposing agencies; these causes usually do not exist in the country and in small communities," etc. The city heat subjects are attended by private physicians only in emergency, being usually summoned to the relief of a man "who has fallen down in a fit while at work." These cases are, therefore, bled—hence, Dr.

Pepper's cases were all bled before admission—and sent to hospital. The majority are admitted between the daily visits of the chief, and so are practically only observed by the resident physician, "who in the discharge of his multitudinous duties" takes no particular notes of these cases. It follows that there is little and vague literature. Dr. Lente reports that in 1847 thirty-seven cases occurred in four days, most of them dying so promptly as to be attended only by the coroner. Animals, omnibus horses especially, were also stricken. Of *forty-two* cases admitted into the New York Hospital in the five preceding years, *twenty-four* died, and eighteen survived. In eight post-mortems there was congestion of the lung in two, and of the brain in four. There was no evidence of inflammation of the brain. One of the most discriminating observations of this paper is that the prognosis depends upon the stage in which the case is seen. So that one "physician might have ten cases, and all might recover; another might have the same number, and the treatment be equally judicious, yet nine out of the ten might die."

In 1852 Dr. C. A. Gordon,[1] an English army officer, had under his care in India, a number of cases of sunstroke, which he reported in 1857, stating that apoplexy and *coup de soleil* include the same conditions of body. Of the twenty-eight cases of heat effect that came under this author's notice but one recovered. His paper is important as bridging the epochs in one of which the effects of heat were ill-described as apoplexy, while in the other the terms insolation and sunstroke evinced a correct appreciation of the exciting cause in some cases, although both terms are too restricted in meaning to embrace the bodily effects of artificial as well as of solar heat.

Dr. G. S. Beatson, staff surgeon of the first class, English Army, criticises the paper of Gordon, because he stated that apoplexy and sunstroke included the same condition of the body and constituted one disease. Beatson admits the close connection between these diseases, but denies their identity in pathology and in treatment if the latter is to include blood-letting. He inveighs against the latter, and quotes fatality as a sequence, and suggests it to be consequence. He opposes to Gordon's almost certain mortality in all cases of this affection a report of eight to ten cases treated in India, in which but one case was bled, and with fatal result, and the remaining cases which were not bled recovered. The treatment of the latter included the removal of the patient to a cool place, the loosening of his clothes, and effusions of cold water on the head, chest, and epigastrium.

In 1853 Dr. T. H. Bache, resident physician of the Pennsylvania Hospital, as he reports in a discussion of Dr. Levick's paper read before

[1] Medical Times and Gazette, London, 1857.

this College in 1858, attended a number of sunstroke cases—they were mostly fatal (six out of ten cases)—and the post-mortem condition of the *heart was not softened*, as reported by Pepper of the cases he had seen.

In 1854 Dr. H. S. Swift,[1] resident physician of the New York Hospital, published his "Observations on Exhaustion from the Effects of Heat (*Coup de Soleil*)." This paper is important as marking a period of some advance and certainly in knowledge concerning heat effects.

The author states that the sixty cases he reports were admitted to the New York Hospital in the "past summer" were of the class at first called *coup de soleil*, but later regarded by the profession as prostration from heat, "combined, perhaps, with the effects of large draughts of cold water." The city inspector reported for that year 260 deaths from *coup de soleil*, without including many cases designated as "congestion of the brain and the effects of cold water." Dr. Swift finally dismisses this topic, saying, "Deaths from the effects of cold water are not so frequently met with as is generally supposed," and then quotes Dr. Dickson, who (p. 492) scouted the production of heat effects by cold water. As the paper of Dr. Moses is the last one found that refers to this subject by title, so is the paper of Dr. Swift the last to at all consider it, unless reference is made to that of Dr. James Trask, who published an account of "Alarming Effects from Drinking Cold Water after Violent Exertion in Cold Weather—February." The symptoms in this case were similar to those of shock, and in no way resembled those of heat effect. The case would not suggest comment in these pages but for Dr. Trask's closing sentence: "The appearance of this man (the patient) resembled that of most persons suffering from the too free use of cold water in the hot weather of summer; the sudden ingestion of a large quantity of cold water causing congestion of the brain." This sentence is the last expression of this opinion found by the reviewer. Its date of publication is 1859, twenty-eight years subsequent to 1831, when the last entry of "effects of cold water" appeared on the records of the Pennsylvania Hospital, where this curious error in diagnosis originated in 1769.

Of the sixty cases reported by Dr. Swift, eleven occurred in a hotel laundry, and several in a sugar refinery. Eight of the sixty cases were women, who, as a class, are unexposed to the sun. These data confirmed the view of Dr. Condie suggested to him by a case occurring in a heated room in the winter of 1818, and the report by Dr. Geddes, in 1846, a case originating in a hospital. They rendered untenable the opinion of Dr. Dowler that the direct rays of the sun (p. 496) were

[1] Swift. The New York Journal of Medicine and Collateral Sciences, 1854, n. s., vol. xiii. p. 45.

essential to pathological results from atmospheric heat, and, moreover, created a need for new and precise terminology descriptive of heat effects in man.

The phrases current at this time for heat effect were apoplexy from heat, *coup de soleil*, sunstroke, insolation, ictus solis, erethismus tropicus, heat exhaustion, nervous debility from heat, and the terminology of Dowler (p. 496). Russell (p. 494) was the first to publish (1836) his knowledge gained by autopsy, that the major form of heat effect was not apoplectic and was coincident with engorgement of the lungs. Dowler, independently, reached the same conclusion by clinical observation (1836), confirmed it later by post-mortem section, and in 1841 published a differential diagnosis between apoplexy and heat effect. He at this time seems to have stood alone in the combined knowledge of the following data:

1. That heat effects were not associated with apoplexy.
2. That they were accompanied by pulmonary engorgement.
3. (*a*) That they consisted of two principal, widely differing varieties, one of "exhaustion" and the other of "asphyxia," or, as would be said to-day, of fever—which he was the first to recognize thermometrically (1845); (*b*) that the treatment of these conditions was in strong contrast, blood-letting being contraindicated in cases of exhaustion, and advisable in selected cases of asphyxia or fever; (*c*) that there was an intermediate and sometimes sequent condition which he respectively recognized under the titles of "pain" and of "excitement or inflammation," and which are now grouped under the single title of meningitis. His predecessors and for twenty years subsequent to 1841, the date of publication of his first paper, his contemporaries confounded the effects of heat with apoplexy. Thus, some authorities maintained that cerebral congestion was always present in heat effect and always demanded depletion. Others held that nervous exhaustion was the only pathological result from heat, and that blood-letting was never permissible. Others again recognized that effects of heat might be exhaustive or congestive, but went astray by identifying fever with apoplexy, cold-water effect, etc.

This confusion of ideas resulted in a babel of tongues, so that *coup de soleil*, insolation, etc., were used by different authors with varying significations and limitations of application. The paper now in review, that of Dr. Swift, typifies this chaos of thought and nomenclature as well as marking, as already noted, a period in which speculation began to crystallize into precise knowledge.

The following quotations from Dr. Swift, as well as those from Dr. Pepper's paper (p. 500), illustrate the contention and misapprehension prevalent on this topic at that day:

"It is now only five or six years since the nature of this disease was

pointed out, and yet the profession, generally, have but vague and indefinite ideas respecting it, and it is a matter of surprise that medical literature is so deficient on the subject."

As evidenced by the references in the foregoing pages, and they are but a suggestion of the publications on this subject, the quantity of literature was not so much at fault as the availability of knowledge of its existence.

Again: "The term *coup de soleil* as applied to this disease is a misnomer. It is a popular rather than a professional appellation. All our authors agree that 'cerebral apoplexy' is occasionally produced by exposure to the direct rays of the sun. This I regard as true *coup de soleil*. The subject now under consideration is an entirely distinct affection. It is now almost universally admitted to be mere nervous exhaustion produced by protracted and violent exercise in an overheated atmosphere."

"Of the large number of cases observed by me, none were strictly apoplectic, and no lesions were noticed in those which proved fatal, sufficient to account for death. These two opposite conditions—the 'cerebral congestion' and 'nervous debility'—require opposite modes of treatment, and should be carefully distinguished from each other." The "cerebral apoplexy occasionally produced" by the sun was current belief. "Of the large number of cases—60—observed 'by him' none were strictly apoplectic," is definite knowledge gained by investigation; nevertheless, current belief, and "all our authors" are not repudiated—but evidence is presented to that end. The reference to "the subject under consideration being mere nervous exhaustion" would mislead to the belief that the minor degree of heat effect as described by Dowler and observed to-day was meant by the author, did he not quote Dowler's description in contrast to his own, and give a most discriminating and detailed symptomatology dividing the condition in mind into three stages: 1. That of onset. 2. That of the course, in which "the skin is hot," "the temperature 112° F.," "according to Dowler." 3. The stage of collapse; this leaves no doubt in the reader's mind that the major form of heat effect is classed under the title of nervous exhaustion.

In a well-marked case that had been classed as having apoplectic symptoms and was bled, death occurred in twenty-one hours. The autopsy, performed eighteen hours later (too late to permit reliable observations—reviewer), discovered no marked congestion of brain or lungs, and a flaccid heart filled with fluid blood. The liver was much congested, other organs healthy. He comments as follows:

"This case was doubtless one of 'nervous exhaustion,' a condition so often mistaken for and associated with 'cerebral apoplexy,' and it was the only one in which reaction ran sufficiently high to indicate depletion. But even in this the post-mortem disappointed us. I have

only seen a few, a very few, cases of insolation verified by post-mortem examination—certainly, not *one* during the past year, although examinations were made *in all the cases* in which we suspected any cerebral lesion."

The following paragraph notes a radical change in treatment: "Blood-letting is now nearly abandoned. Formerly nearly every case treated before admission to the hospital had been bled. But not a *single patient* had been bled of those admitted during the past summer. They do not bear well even the local abstraction of blood by cupping."

It was the period of reaction. Before laying down this very interesting paper, it will be well to note some of its statistics: The total number of cases admitted to the New York Hospital from 1845 to 1854 was 150, of which 78 died. The number of cases under Dr. Swift's observation was 60; the mortality more than one-half. On admission 44 cases were insensible, 16 stupid or sensible; the pupils were dilated in 30, contracted in 19, and natural in 11. The temperature of the body was hot in 34, warm or natural in 11, cool in 18; respiration hurried in 44, pulse uniformly accelerated 100 to above 160; convulsions in 24; delirium in only a few; 52 were males; and the average duration of fatal cases about four hours. "In 33 fatal cases the pupils were contracted in 20, moderately dilated in 7, and markedly so in 6; while in the successful ones the pupils were dilated in 19, and nearly natural in 16. No case recovered in which the pupils were contracted. The treatment included the hot bath, sinapisms, enemata of aloes or turpentine and "ice to the head *when the temperature is elevated.*" . . . "Indiscriminate use of cold affusions is productive of harm. Injurious and often fatal effects result from them."

In 1854 Dr. J. R. Leaming, in the same number of the *New York Journal* as the paper of Dr. Swift (p. 58), advises for heat effects the use of hydrochlorate of ammonia in 8-grain doses. The suggestion is made from the employment of the drug in four cases concerning which the author truly states his notes to be "very meagre."

In 1856 Dr. Sanford B. Hunt[1] published his "Observations on the Cause of the Disease Known as Sunstroke." In this paper, dealing largely but with greater originality and aggression with the facts observed concerning the large number of cases occurring in the service of Dr. Swift, in 1854, reactionary opinion swings further, and in some instances the conclusions are broader than the facts.

He first considers the question:

Are the direct rays of the sun necessary to the production of the disease known as sunstroke?

[1] Sanford B. Hunt. Buffalo Medical Journal, 1856, vol. xii. p. 23.

He cites that in the sunstroke *epidemic* in New York, August, 1854, of the 235 deaths from this cause, 49 were females. In view of this fact and the comparative non-exposure of females to the sun, added to the occurrence of the laundry and sugar-house cases, and of similar cases, as recorded by the health offices of various cities answers this question, beyond contention, in the negative.

Secondly, is congestion of the brain the special pathological condition in this disease?

Again referring to the cases of Swift, Dr. Hunt says: "At any rate, enough is known to prove that contracted pupils and convulsions are not to be accepted as sufficient proof of congestion, and we must wait for actual post-mortem evidence of it to verify its existence. It is already plain that only a very limited number of cases are congestive, and it is quite probable that that limited number will be much decreased on careful study."

He approves Swift's title of "Nervous Exhaustion from the Effects of Heat" as the designation of the disease, and immediately beneath the second inquiry of the paper says: "It is only recently that the fact has been fully recognized, that in the great majority of instances of sunstroke, the symptoms have been those of syncope or exhaustion." Knowledge of the paper of Dr. Swift and of the fact that he includes under the title of exhaustion major cases of heat effect, make it plain that Dr. Hunt like Dr. Smith used this term in error, because too broadly denominating by it, heat effects of hyperpyretic character as well as those of exhaustion from heat to which the phrase should be limited as suggested by Dr. Dowler and practised to-day.

What are the facts in relation to heat as a cause of coup de soleil?

He answers this question by quoting meteorological data in evidence of a greater number of cases of heat effect occurring on hot humid days than on hotter days but of dry heat.

He states: "The facts adduced are conclusive that, though a certain temperature is necessary, neither the frequency or the fatality of the disease increase with a further rise of the thermometer. Heat is not the essential cause; neither are the direct rays of the sun necessary."

"What then is the essential condition for the production of *coup de soleil*, or rather exhaustion from the effects of heat?"

"High humidity is the essential condition. This opinion is based upon weather records and upon analogy."

"In the first place the condition of nervous exhaustion is never produced by a high, dry heat."

Dr. Hunt disclaimed originality for his views on the etiological influence of the disease "known as sunstroke," yet such a claim would have been pardonable, considering that he is the first to present the

scientific evidence of hygrometry in support of an observation probably first published by Lancisi, as already noted in 1694. It hardly is necessary to say that the influence of humidity in the production of heat effects is generally acknowledged, but when Dr. Hunt says that they are "never produced by dry heat"; "that, though a certain temperature is necessary, neither the frequency or fatality of the disease increase with a further rise in the thermometer;" and finally, "that the essential condition for the production of *coup de soleil* is high humidity and not heat," he goes too far. The paper, as a whole, is one of much originality, force, and some discovery; it finally refers to the institution of "experiments to determine as to what power a humid atmosphere exerts on the absorption of the solar rays," and the message of the author is best expressed in his sentence saying, that "examination of such weather records, corresponding with periods of mortality from sunstroke, as have fallen under 'his' notice, have uniformly resulted in fixing a high temperature of evaporation as the efficient condition of the cause of the disease, and that without definite relations to the dry bulb temperature."

In 1856 Dr. J. B. S. Jackson[1] reported a case of recovery from sunstroke which occasioned discussion in which Dr. Strong presented a classification of cases into two groups not very clearly described.

Drs. Parkman, Cabot, Bigelow, Senior, and Coale also took part in the discussion.

In 1856 Dr. Charles Morehead,[2] in the first edition of his *Researches on Disease in India*, treats of the pathological effects of heat under the headings of "Apoplexy" and "Cerebral Affections Caused by Elevated Temperature."

In his consideration of apoplexy he at once distinguishes between a "correct" use of the word as signifying cerebral *congestion*, with or without effusion or hemorrhage, and a looser use of the term, including in its meaning cerebral "*determination*" as applied to the effects of excessive heat or of alcohol. He quotes statistics in evidence that heat effects were often and erroneously classed as apoplexy.

In treating of "Cerebral Affections Caused by Elevation of Temperature" he makes a triple classification of symptoms, which, it is plain, are those of three stages in the major form of heat effect. He records that the pupils in the cases he observed were primarily contracted, but finaly dilated—an observation later confirmed by Dr. Stiles' experimental investigations.

The pathology noted includes vascular turgescence of the membranes

[1] J. B. S. Jackson. Extracts from Records of Boston Society for Medical Improvements, 1856, vol. ii. p. 9.

[2] Morehead. Clinical Researches on Disease in India, London, 1860, first edition, vol. ii. pp. 577–582.

and substance of the brain, posterior congestion of the lungs, engorgement of the venous circulation and of the right heart with badly coagulated blood. Phrenitis and encephalitis are pronounced of rare occurrence.

The following paragraph presents a "suggestion" that has claim to priority, and that anticipates some of the conclusions derived from experiments reported three years later by Bernard:

"Active cerebral determination of blood takes place as a consequence of exposure to elevated temperature, and coexists with the symptoms above detailed; but it may be doubted whether it is a sufficient explanation of the proximate cause of the phenomena. From a review of all the attendant circumstances it seems to me not an unreasonable suggestion to offer that *the temperature of the blood may become much increased, and to this altered condition of the blood the deranged actions may in part be due.*[1] The nature of the cause and the pungent heat of the surface of the body, which is invariably present, may be used as grounds for this opinion. The tumultuous action of the heart is also a characteristic symptom, and seems to me to point to a direct influence of the *cause* on that organ."

Although Dowler was the first to measure the degree of elevation of temperature, he, like Russell, attributed the chief cause of fatality to the lungs. Morehead was the first to suggest the abnormal elevation of the temperature of the blood to be the primary cause of trouble in heat effects, and its *direct influence* to be secondary.

In 1857 Dr. B. Darrach,[2] resident physician in the New York Hospital, treated four cases by stimulants and by *rubbing the entire surface of the body with ice;* three of his cases recovered, one died. He reports the cases equal in severity to four previous cases in which the ice was only applied to the head, and all four patients died. This is the first report of the application of ice to the whole body in the treatment of this condition.

In 1858, Claude Bernard[3] announced in his lectures to the College of France that the temperature of mammals and birds could be elevated 4° C. to 5° C. (7.2° F. to 9° F.) above normal, and that the animals, without exception, died when their temperature became elevated to this degree. On autopsy, the *heart* and other muscles of the body were found *rigid.* In the history of the pathology of the major form of heat effects, this communication is only second in importance to the first observation of temperature in this disease, by Dowler in 1845. The significance of the phenomena observed by Bernard will become more apparent with the reading of his report of further experimental data and of his deductions.

[1] Italics introduced.
[2] Darrach. New York Journal of Medicine, 1854, n. s., vol. xii. p. 45.
[3] Bernard. Lectures to the College de France, 1858, vol. iv. p. 209.

In 1858 Dr. Levick[1] reported to the College of Physicians twelve cases treated that year at the hospital; one of these cases occurred in a sugar refinery, three cases died, and also a cured case suddenly on the sixth day after recovery. Dr. Levick notes the differences between exhaustion and congestion as bodily results of heat, and urged that for these dissimilar conditions different modes of treatment were required, and that, therefore, diagnosis was important, and he italicizes his belief that the process is most frequently one of exhaustion. He deemed none of his cases in need of bleeding. He comments upon the coincident value of turpentine in the treatment of malignant fevers and of sunstroke. He notes the post-mortem appearance of the blood in typhus to resemble that in sunstroke; that putrefaction in the latter disease was so rapid as to prevent autopsy ten hours after death; that in the last case reported symptoms of inflammation of the brain were corroborated by post-mortem discovery of extensive inflammation of its base.

In January, 1859, Dr. Levick[2] contributed to THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES the most comprehensive history and analysis of this subject published at that time, and to the references of which this review is much indebted. In confirmation of artificial and solar heat being alike productive of the same symptoms, as evidenced by the cases reported by Swift, Dr. Levick alludes to the case already noted, which he reported the previous year, and which developed in a sugar refinery. Contrary to first impressions, he subscribed to the observations of Dr. Hunt with regard to humidity being an etiological factor "of the disease known as sunstroke," but Dr. Hunt's assertion that disease from heat is "never produced by a high, dry heat" is disproved by Dr. Levick, who quotes the case detailed by Russell as occurring "at a season when the hot land winds had just set in, rendering the atmosphere dry and suffocating."

Dr. Levick, like Andral, comments upon the coincidence of atmospheric heat, and the epidemics of apoplexy noted by Forestus, in 1562; but while Andral would confirm this observation, Dr. Levick maintains that the epidemic character of the disease in hot, humid weather is only explainable by the inference that at least many of the cases were *not* due to apoplexy, but to the effects of heat.

He notes and accepts the post-mortem observations that establish apoplexy and heat effects to be dissimilar conditions, and also those concerning the fluidity of the blood and the general venous and especially pulmonary engorgement. He again comments upon the rapidity of putrefaction noted by him the year before. He quotes Pepper, Lente, and Swift in agreement upon the flaccid condition of the heart.

[1] Levick. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1858, vol. xxxvi. p. 404.
[2] Ibid., 1859, vol. xxxvii. p. 40.

He by inference condemns blood-letting. He advocates cold affusions and the use of ice to the head in selected cases. He warns that "the full bath is not without danger." The chief indication for its use is found in the stimulus afforded by the *hot* bath. He finally quotes the "happy results" obtained by Dr. Darrach in "frictions of the body *with ice*" to relieve the burning heat of *the skin*, which is justly viewed as a most unfavorable prognostic symptom. Carbonate of ammonia and the aromatic spirits are recommended for administration by the mouth, and oil of turpentine by the bowel.

This very exhaustive article closes with the following reference to sequelæ which are best presented in the words of the author, as future reference will be made to it:

"After the immediate symptoms of an attack have passed, as after cholera, typhus fever, etc., so with sunstroke, various internal inflammations may be developed—as of the brain, the lungs, etc.—which require a treatment entirely unlike that mentioned above. In this way we have cases reported as meningitis resulting from exposure to heat; but according to my observation, these differ from sunstroke in their gradual mode of attack, although just as apoplexy may occur in warm weather, so may meningitis; but in by far the greater number of cases of sudden attacks of illness from exposure to heat, inflammation of the brain is not the pathological condition. Nor must we be misled by the presence of delirium, or of a hot skin, in such cases. The latter we have present even in malignant typhus, and no fact is better known to the pathologist than this; that, in typhus and typhoid fever, phenomena almost identical in character with those of inflammation of the brain may be present, and yet, after death, no lesion of this organ be found. In the words of Dr. Bartlett, when writing of typhus fever, "delirium and somnolence have occurred as frequently, and as strongly marked, in patients whose brains presented no changes, as in those of an opposite character."

It is interesting to note that general frictions with ice were approved by the author for the relief of an "unfavorable *symptom*" affecting the skin and employed empirically. The pathology demanding refrigeration as one of the vital principles in the treatment of the major form of heat effect was not yet understood. That it had been divined, is suggested by the comment of the reviewer (p. 509) on the notes of Bernard. His second announcement, now in order, sheds much light on the pathology of heat effects, implying the essential necessity of employing cold in their treatment.

In February, 1859, Claude Bernard[1] reported the results of two experiments "on the cause of death in animals submitted to a high

[1] Comptes-Rendu des Séances et Mémoires de la Société de Biologie, 1859, Tome iii. Series B. 51.

temperature." He refers to his announcements of the year before and presents the data of subsequent observations and his conclusions.

Experiment 1. A rabbit subjected to a temperature of 55° C. to 60° C. (131° F. to 140° F.) fell on its side and died quickly. The temperature of the rectum and thorax taken at once was 44° C. to 45° C. (112° F. to 113° F.). The heart was still, was firmly contracted, and contained no blood. The muscles and limbs were stiff, and did not respond to galvanism. The blood of the ears was black, presenting no apparent alteration of character. Poured in a glass it coagulated well, and the exposure exhibited a ruby red color.

Experiment 2. A younger rabbit that had not been permitted to feed preceding the experiment, as had the first rabbit, succumbed a little more slowly to the effects of heat; otherwise the same phenomena were exhibited at both experiments.

He explains the cause of death in these cases to be *due to the purely physical fact of the elevation of the temperature of the blood.* This elevation is such as to be incompatible with the exercise of muscular contractility, and consequently of the cardiac functions. The heart is stopped and becomes incapable of vital functions. The muscular fibres of animals having a precisely similar temperature exhibit after death rigidity and immobility. Dr. Kühne has shown that in these conditions the rigidity was due to coagulation of a special matter contained in the muscle. As he had observed the muscles of animals dying of cold, although rigid, to be of alkaline reaction, he concludes that the usual muscle acidity accompanying cadaveric rigidity should not be considered as a cause of rigidity, since the latter can exist without acidity.

In 1860 Dr. Charles Morehead[1] published a second edition of his *Clinical Researches*, containing an enlarged chapter on "Sunstroke." This title, condemned by the author in his first edition, is employed in the second, and broadly applied to the effects of the indirect as well as the direct rays of the sun. This chapter represents the experience and opinions, unpublished as well as published, of many Anglo-Indian surgeons as well as the personal observation and views of the author. It contains a new classification of symptoms dependent upon an Anglo-Indian recognition that in the effects of heat there are different varieties, which he denominates as follows:

1. Comal, which is cerebro-spinal in origin.
2. Syncopal, which is a cardiac type.
3. Comal and syncopal combined, the origin of which is cerebro-spinal as well as cardiac, and is therefore denominated as "mixed."

The symptoms detailed of the first, or cerebro-spinal variety, show it to be identical with the condition known to-day as thermic or heat fever.

[1] Morehead. Clinical Researches on Diseases in India. London, 1860, second edition, p. 603.

The second, or cardiac variety, may be subdivided into two types. In one the individual, "without premonitory warning," and generally consequent on direct exposure to the sun, falls down, gasps a few times, and dies.[1] The authority for this instantaneous form is assigned to Dr. Pirrie. Such instant fatality is contrary to the experience of most observers. Rush described it, but his contemporaries Condie and Watt discredited it. Dowler reports instances of death occurring in twenty or thirty minutes, but hours are usually occupied in the fatal progress of this affection. The second form of the cardiac variety is not instantly or necessarily fatal. This subdividing of the cardiac variety is a distinction made by the reviewer, and not the author. The symptoms detailed of this type—for instance, "a cold and clammy skin"—identify it, without doubt, as that of heat exhaustion, in spite of the fact that the author groups in it cases of most unusual and instant fatality.

In the third, or mixed variety, the author assembles atypical cases of the two varieties described, more especially mild cases of the first variety.

On the topics of symptoms, pathology, etiology, and treatment, the author, though at times in disagreement with modern conclusions, often penetrates with clear-minded energy to first causes or perceives indications that were not recognized as true by many observers until years later. Justice can only be done this most instructive chapter by consulting the original.

In 1863 Dr. Horatio C. Wood, Jr., published in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES a report of eight cases affected by heat, seven of which died; a ninth case that died is recorded in the hospital records, and was probably not under Dr. Wood's care. He expresses his belief that heat produced different classes of cases called sunstroke, but he had observed only the fatal variety which he had reported. Dr. Wood is the first of the staff of the Pennsylvania Hospital to publish thermometric observations of his cases, but the first reports of this character are those of Dr. Dowler. The maximum temperature observed by Dr. Wood during life was 109.75° F., but one hour after death the temperature of 110.75° F. was recorded in one case.

Dr. Wood's report is still unique in its wealth of post-mortem notes. In the six autopsies recorded the following observations were most constant. None but the slightest congestion of brain, meninges, and lungs, although the meningeal and pulmonary veins were loaded with blood. The blood was dark, fluid, tending to acid reaction and not to coagulate. Its microscopical examination in one case is the first recorded in this disease. The red globules were found "a little more

[1] Pirrie. Insolation, Sunstroke, or Coup de Soleil, Lancet, May 20, 1859.

opaque than normal, and aggregated in rouleux and masses. No abnormal proportion of white corpuscles." "The *heart* was in all cases *rigidly contracted*. The veins were surcharged with blood." In this, as in other diseases, ecchymoses are characteristic of altered blood. The pathognomonic post-mortem results of fatal blood disorders are like those of sunstroke; "alteration and destruction of the crasis of the life fluid, without structural changes in the solids."

He states that alterations in the blood produced by heat are responsible for the cerebral symptoms observed, that death is brought on by loss of power in the central nervous system, and he substitutes thermohæmia, or "thermic fever," as the name of the condition in place of sunstroke. The name thermic fever is an advance in specific terminology, because the first word accurately gives the cause of the trouble, and the latter word states the condition resulting from the cause; the two words combined limit their application to the grave form of bodily effects from heat which is accompanied by an elevation of temperature that tends to the extreme. He condemns the prejudice against drinking water, and urges the ingestion of fluids, because the blood has been deprived of its serum, it may be by pints, through the action of the perspiratory glands. He acknowledges treatment to have been futile in his hands, and quotes and suggests Taylor's[1] recommendation of cold affusions as practised in India.

An analysis of Dr. Wood's reports of his cases discovers that cold was employed only in the treatment of three, namely: Cases III., IV. and VI. Cases III. and IV., axillary temperature 109° F., had ice frictions; Case VI., femoral temperature 104° F., was treated with cold affusions, *which aggravated the tendency to convulsion*. Two of the cases were intemperate; the other, temperature 109° F., was sixty years of age. The two cases of 109° F. were not brought to the hospital until two hours after the onset of the attack. The cases of 104° F. had been suffering from the effects of heat four days before admission. These were all unfavorable cases for treatment, and they all died.

In 1864 Dr. R. Cresson Stiles[2] published the results of the first experimental "observations on animals subjected to an elevation of temperature in various modes, and which presented a series of symptoms very closely allied or identical with those of sunstroke as 'he' had seen it, and as 'he' found it described by careful observers." These experiments were inspired by the diversity of existing opinion respecting sunstroke, by a desire to test the accuracy of the "currently received pathology of the disease," and with a prejudice in favor of a poisonous quality being acquired by the blood. This theory he credits

[1] Lancet, 1858.

[2] Boston Medical and Surgical Journal, 1864, vol. lxx. p. 349.

to Dr. H. C. Wood, in the publication just summarized. The investigations of Dr. Stiles soon caused him to abandon this theory or rather to "regard the only poisonous property of the blood to be its elevated temperature."

Dr. Wood, in his *Boylston Prize Essay*, 1872, p. 103, thus concurs with Dr. Stiles: "I believed at that time (referring to his paper of 1863) that the nervous symptoms were due to the development of a poison in the blood;" not perceiving that the heat itself was the poison.

His conclusion is formulated in the title of his paper: "Sunstroke, or Paralysis of the Heart by Heat."

He reached his conclusions by five classes of experiments:

Class 1. "Those in which warm-blooded animals were subjected to an elevation of temperature; the symptoms they manifested observed, and post-mortem appearances noted."

Method. The animals were either placed in glass bell-jars of sufficient capacity for free respiration—and the jar covered and immersed in water of 115° F., later raised to 120° F.—or, held by the nape of the neck, they were directly immersed in the heated water.

Results. They exhibited the phenomena of sunstroke, and died. A hearty meal hastened fatality. The sequence of symptoms were restlessness, increased pulse, and respiratory rate, distention of bloodvessels, slight outcry, coma, primary dilatation of pupil, soon followed by its contraction. Upon contraction of the pupil, subjection to artificial heat was discontinued. Within a half-hour of the occurrence of pupil contraction, the bloodvessels cease to be distended, then cyanosis, slowing of the respiration, convulsions occasionally, and death follow. The animals directly immersed in water exhibited within five minutes rigid and permanent contraction of all muscles.

Autopsy, of immediate performance, discovered abdominal and thoracic temperatures not above 115° F., and either the heart ventricles firmly contracted, or permitted the observation of their contraction within ten minutes after death. The auricles were distended with blood and exhibited fibrillar movements. Neither brain nor lungs were congested. The venæ cavæ were engorged with dark alkaline blood rapidly coagulating. The cut heart ventricles were acid in reaction. The microscopical appearance of the blood was normal, but that of the heart fibres showed loss of striæ. In a few hours heart rigidity gave way to flaccidity. Putrefaction was soon manifest. Hence, the prompt autopsies made by Dr. Horatio Wood discovered heart contraction, in contrast to the flaccid hearts observed several hours after death by Dr. Pepper.

Class 2. "Those in which artificial respiration was practised as soon as the animal became comatose, or the pupil became contracted."

Results. Life was not thus prolonged even subsequent to tracheotomy.

Class 3. "Those in which blood heated to 120° F. was injected into the veins of the animal from which it had been drawn, or in which the blood of an animal perishing from the disease produced by heat was injected into the veins of an animal of the same species."

Results. No toxic symptoms ensued.

Class 4. "Those in which it was proved that the muscular system of an animal could be paralyzed by heat while its nervous system was left intact."

Method. "The reflex actions of animals apparently moribund from the effects of heat presented a decided contrast to those of narcotism, or exhaustion of the nervous system." In cold-blooded animals, on account of the sluggishness of their circulation, the heart is the last organ to be influenced by a heated liquid. When thrown into water of 115° F. all the muscles of a frog become rigid in a few minutes. In partial immersion only the part immersed is affected. After immersion and rigidity of long duration, not only do irritation of distinct nerve centres cause convulsions of the immersed parts, but irritation of nerves locally and equally exposed to the heat that has rendered the muscles of the part rigid will meet convulsive response.

Hence the nervous system is less sensitive to heat than the muscular.

Class 5. "Experiments on muscular contractility is affected by heat."

Results. The more violent the activity of muscles subjected to heat, the more promptly did they become rigid. While the cut surfaces of muscles made rigid by heat are decidedly acid, those of animals dying of other agencies (*e. g.*, section of medulla or toxic tetany) are not. The non-striated muscular fibre of arterioles of heat-stricken animals would not contract in response to cold or galvanism, but intestinal peristalsis was well marked. The normally contracted pupil of the dead frog dilated on immersion in water at 118° F.

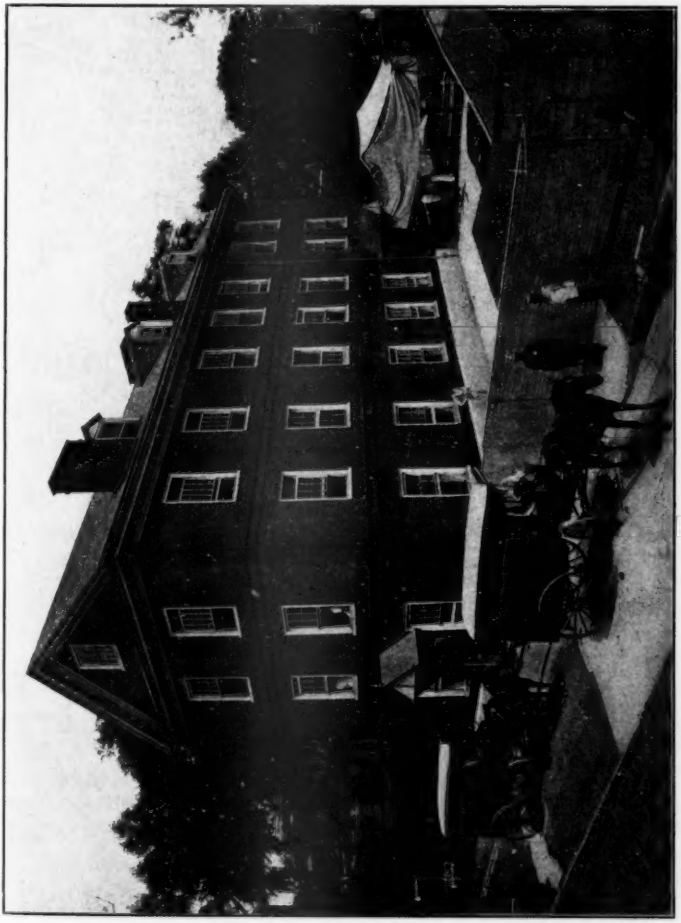
The experimental data of the paper was supplemented by clinical and post-mortem observations on man, and a presentation of existing theories of his own. Finally, in the recapitulation of his experimental results are the following notes:

"A temperature of the blood below 115° F. is sufficient to destroy life in all vertebrates, and causes phenomena similar to those of sunstroke, while a temperature between 115° F. and 120° F. is sufficient to cause rigid contraction and paralysis of the muscles whether connected with or separate from the body of the animal. The more violently a muscle is made to contract before subjection to heat the sooner it becomes paralyzed."

Dr. Dowler's observation of axillary temperature of 112° F. establishes that the temperature of the blood in sunstrokes is sufficient to produce paralysis. The dilatation of the capillaries is explicable by the direct effect of the heated blood upon the muscle fibres of the walls of the arteries and arterioles. The cerebral symptoms and the full and forcible pulse may be due to this dilatation.

"The effect of heated blood upon the heart is termed paralysis rather than exhaustion, because the heart has been shown to have the power of directly paralyzing its muscular tissue, and because in sunstroke the first stage, or that of frequent and forcible contraction of the heart, is not an essential part of the disease."

This paper not only confirmed the announcements of Bernard, although it does not allude to them, but it also contributed much new knowledge which later was largely corroborated by other observers, and doubtless helped to inspire their work.



Receiving ward (North House) and sunstroke tent, Pennsylvania Hospital.

In 1866 Dr. George B. Wood, visiting physician to the Pennsylvania Hospital, in the sixth edition of his *Practice of Medicine*, advocates the term *heat fever* in preference to the term *thermic fever*, suggested by Dr. Horatio C. Wood, and of which heat fever is but a translation. Either of these names Dr. George B. Wood prefers to insolation, etc.,

because the condition in mind though a consequence to heat is not one exclusively of solar heat.

In 1866 Dr. Levick, as he reports in 1868, treated 22 cases in one week at the hospital (see Graphic and Statistical Chart, in later publication). Of 12 cases treated by affusions and entire bath, 7 died. Of 7 rubbed with ice, 6 recovered; 2 of these had temperatures of 109° F. This is the first record of a recovery from such elevation of temperature, and it apparently marks the first fully intelligent application of cold in the treatment of heat fever. The earlier use of general ice frictions (p. 511) was to relieve the heat of the *skin*. The treatment by Dr. Levick of the cases occurring in 1866 must have been instanced by his appreciation of the indications of the newly discovered pathology of this disease, for general refrigeration of the blood, and, with equal propriety, it might be said of the muscular tissues, but that Dr. Levick in the paper next in order of review, refers only to that portion of the paper of Dr. Stiles that deals with the blood, and makes no allusion to his discoveries concerning the action of heat on muscle.

In 1868 Dr. Levick, in the *Pennsylvania Hospital Reports*, vol. i., p. 24, contributes a paper entitled, "Heat Fever (Sunstroke)," and credits the title of Dr. George B. Wood. No allusion is made to "thermic fever," a title of prior date, coined by Dr. Horatio C. Wood.

The paper begins and ends with emphasis on the fact that "two or more pathological conditions, very distinct in their phenomena if not in their nature, are often spoken of indiscriminately as insolation or sunstroke." It has two "obvious forms," and another, of which he says, "surely, it were an unnecessary refinement to give this disease a classification separate from meningitis." He, therefore, differentiates, and so clearly as to need no present modification, three forms of heat effect: 1. *Exhaustion from Heat.* 2. *Meningitis.* 3. *Heat Fever.* Dowler, in 1841, made the same classification with a different terminology, limiting the exciting cause to solar heat; whereas, Dr. Levick states specifically that either of these forms of heat effect may arise from solar or from artificial heat. He also refers to high humidity as a strong etiological factor in the production of pathological results from heat, and suggests that the mortality on the "Liverpool" in the Persian Gulf was possibly increased by keeping the decks wet (p. 491). Due credit is given the paper of Dr. Hunt.

A detailed clinical history, with notes of autopsy, of microscopical examination of the blood, and of a control experiment with, and examination of, the blood of a healthy individual, is followed by a claim for priority in noting the resemblances between the "phenomena of sunstroke and those of an idiopathic fever." The claim is supported by quotations from the essay on sunstroke in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1859, p. 50, already noted and by a repe-

tition of the parallel between the symptoms and post-mortem appearances of heat fever and of typhus fever as a type of the idiopathic fevers. The teachings at the Pennsylvania Hospital of Drs. Gerhard and Pepper that the "essential pathology of this disease (the major form of heat effect) was to be found in an altered condition of the blood" is cited. "It was but a step further to notice the resemblance of the phenomena to those of an idiopathic fever."

The microscopical examination of the blood is the third reported in this condition, being preceded by that of Dr. H. C. Wood and also by that of Dr. Stiles in his experimental work. The negative observations of Dr. Stiles are in agreement with those of Dr. Wood and of other investigators of the appearances of the blood in this condition. Dr. Levick stands alone in reporting crenation of the corpuscles. The control experiment was made at his request by Dr. Roland G. Curtin, of the Philadelphia Hospital. Of four ounces of blood taken from a healthy woman, and divided into two equal portions, one was microscopically normal and clotted in twelve minutes. The other was gradually heated in a sand bath until the blood reached a temperature of 120° F. "The second portion was gradually heated in a sand bath until the thermometer placed in it was at 120° F. Under the microscope, the red corpuscles were now found to be about half their usual size, shrivelled, and crenated. This blood coagulated in five minutes less time than the first portion did. So far then as this proves anything, it would seem to show that while the vitality of the corpuscles of the blood may be destroyed by heat alone, something more than this is required to destroy its property of coagulating, when drawn from the circulation.

In the *Essay* of 1859, p. 51, already quoted on this point (p. 510 of this review), Dr. Levick expressed his belief in a "poisoned condition of the blood." The following quotation, referring to this point, shows a modification of opinion: "Absence of toxic properties would seem to be established by the experiments of Dr. R. Cresson Stiles." . . . It is interesting to recall that Dr. Horatio C. Wood expressed the same belief in 1863, and, as already stated, recants it in 1872.

The question of change in theory in treatment indicating the application of cold was raised on page 511 in noting the cases occurring and treated in 1866. The following paragraph presents Dr. Levick's view on this subject:

"Of the *modus operandi* of ice in these cases, but little need be said. In all probability it acts by its refrigerating properties alone, and yet, when the instantaneous effect of a drop or two of water on the red corpuscle as seen under the microscope is recalled, the question will present itself, Can there be any endosmotic action of the water in these cases which is eagerly seized upon by the parched blood?"

This third paper of Dr. Levick is of epochal import, and marks the beginning of intelligent treatment of the conditions considered.

In 1868, in the second volume of the Hospital Reports, Dr. James Hutchinson publishes a paper upon the success of one-fourth grain, hypodermic doses, of morphine in the control of convulsions in four[1] cases of sunstroke treated at the hospital that year. The injections were suggested and given by Dr. Herbert Norris, resident physician.

In Period IV., 1841–1870, 164 cases of heat effect were received at the hospital, the mortality being forty-nine. In this epoch marked progress was made in the study and treatment of heat effects. The most notable events were the first use of ice-water affusions, by Markoe, and of ice applied to the head, as noted by Dowler, both in 1841; the publication by Dowler, in the same year, under a different terminology, of the classification recognized, but not published, by Condie in 1818; the further classification by Dowler of a meningitic type; the recording by Dowler, in 1845, of the first thermometric observation of hyperpyrexia (113° F.) in the major form of heat effect; the suggestion by Morehead, in 1856, that the pathology of the condition depended on the temperature of the blood being abnormally increased, and possibly acting directly on the heart; the first application by Darrach, in 1857, of treatment by general refrigerating friction with ice; the confirmation of Morehead's "suggestion" by the experimental work of Bernard in 1858–59 and by Stiles in 1864; the comparison of heat effects with fevers by Levick, in 1859; the discoveries of the last two observers of the effects of heat upon muscle and nerve; the negative microscopical examination of the blood by Wood, in 1863, and his denomination of the major form of heat effect as thermic fever; and, finally, the institution of an intelligent use of opium in the control of convulsions by Hutchinson and Norris, in 1868. The achievements of the period reviewed were but preparatory for further advances in the period 1841–1870, which will be presented in a later publication.

[1] In four cases is this use of morphine recorded by Dr. Hutchinson—not sixteen, as stated on p. 110, by Dr. Horatio C. Wood, in Sunstroke.

REVIEWS.

REGIONAL ANATOMY OF THE HEAD AND NECK, FOR STUDENTS AND PRACTITIONERS OF DENTISTRY. By WILLIAM T. ECKLEY, M.D., Professor of Anatomy in Dental Department, University of Illinois, in the College of Physicians and Surgeons, Medical Department University of Illinois, etc., and CORINNE BUFORD ECKLEY, M.D., Demonstrator of Anatomy in Dental Department, University of Illinois, College of Physicians and Surgeons, etc. With 26 plates in black and colors, and 36 illustrations in the text. Philadelphia and New York: Lea Brothers & Co.

IT may be true that there are departments of anatomy with which the dentist is particularly concerned, but it is not true that he is particularly concerned with certain departments of anatomy only. The intelligent practice of dentistry must be founded upon a fair working knowledge of the structure of the entire human organism, and the inference may be readily drawn from the existence of a special work of this character that it contains all that is necessary for a dental practitioner to know of the special subject of which it is the exposition of only a small though even very important portion. We fail to see that this work contains anything of special importance which is not adequately set forth in standard works on the general subject of anatomy. The arrangement of the various topics is illogical in the extreme. The initial chapter is a chapter of forty-one pages, which is devoted to the Anatomy of the Trifacial Nerve, followed by about twenty pages more of description of the muscles of mastication. Chapter III. is devoted to a description of the roof of the mouth, and Chapter IV. to the mouth itself. Why such a division should be made does not appear. The balance of the subject, fairly covering the Anatomy of the Head and Neck, concludes with Chapter XVII. upon the Osteology of the Region.

There are numerous inaccuracies in the work, and a large number of repetitions which mar its usefulness—for example, in stating the composition of the pre-maxillæ, "the corresponding part of the lips is included." It is unusual to describe the soft tissues as part of a bone. Of the ethmoidal cells, he states that the anterior open into the frontal sinus. The published dissections of Cryer show that such is not the case, and further, page 96, with reference to the anterior ethmoidal cells, the author states that they open into the middle meatus of the nose. Page 111, it is stated that the mental artery is a branch of the internal maxillary, while as a matter of fact it is a branch of the inferior dental. Page 115, the statement is made that "The orbicularis oris surrounds the mouth," a looseness of statement inexcusable in a scientific work.

At page 135, in describing the common roof structures of the five triangles of the neck, no mention is made of the platysma myoides.

Whole paragraphs, even pages of text, are repeated at times almost verbatim, and the work will need most thorough revision in order to justify its publication and make it a useful text-book for students. Therevision should comprise not only the elimination of actual errors, but a more orderly arrangement and the avoidance of so much repeated matter.

The mechanical make-up of the work is most excellent. The illustrations, which are almost wholly taken from standard authors, are of a high grade, the paper and presswork being above criticism.

E. C. K.

DIE VERDAULICHKEIT DER NAHRUNGS—UND GENUSSMITTEL AUF GRUND MIKROSKOPOPISCHER UNTERSUCHUNG DER FÆCES. Mit 102 Abbildungen. Von Kreis-Physicus a. D. Dr. Med. F. SCHILLING. Leipzig: Hartung & Sohn.

DIE FÆCES DES MENSCHEN IN NORMALEN UND KRANKHAFTEN ZUSTAND MIT BESONDER BERÜCKSICHTIGUNG DER KLINISCHEN UNTERSUCHUNGSMETHODEN. Von DR. AD. SCHMIDT und DR. J. STRASBURGER, Privatdocenten an der Universität Bonn. I. Theil, mit 6 lithographischen Tafeln. Berlin: August Hirschwald.

CHEMICAL investigation of the feces has given the student of the digestive processes much information concerning the results of intestinal changes, but has been of very secondary value as a help to clinical diagnosis or to treatment. Within recent years, however, since Szydlowski, in 1879, laid the first really substantial foundation for the study of the feces, unceasing effort, in certain lines, has been put forth in the study of the intestinal dejecta. These two works represent the latest and best observations on the subject. Ledder-Hulsebosch, in 1899, presented an admirable work on the same subject, but limited his observations to vegetable detritus; both of the works here mentioned treat of the animal foodstuffs as well.

The former work of Schilling may be dismissed with a few words—it is an excellent résumé of the subject, and from the general point of view would make a good introduction to the study. The figures are numerous, and for the most part good, although inclined to be schematic. His illustrations of the various types of muscle detritus are open, we believe, to criticism. It is doubtful if the differences he notes in the muscle cells of allied animals are as distinct as his drawings would lead one to believe.

The work of Schmidt and Strasburger is, in the first portion here offered, a most excellent one, and deserves special commendation. To the clinician who is keenly alive to the importance of intestinal digestion this work will come as an added help to illuminate this dark chapter of symptomatology. Especially is this true for the pediatrist, and, if we read the signs of the times, a much needed light for the study of the intestinal dejecta, beyond the casual observation and perhaps the use of litmus paper, is, for the most part, done in a most perfunctory manner.

There is no doubt that, for American medicine at least, the use of the microscope as a means of diagnosis in intestinal derangements has been

greatly undervalued, and the more extended application of this aid to clinical medicine would do away with many of the peculiar ideas concerning certain forms of diet. S. E. J.

THE PRACTICAL MEDICINE SERIES OF YEAR-BOOKS. Vol. V. OBSTETRICS. By REUBEN PETERSON, A.B., M.D., and HENRY F. LEWIS, A.B., M.D. Chicago: The Year-Book Publishers, 1901.

IF the whole series of *Year-Books* is equal in excellence to the volume before us, nothing more can be asked. As its title indicates, the aim of the series is to review the advances made in medicine and surgery during the past year. This, in the present volume, has been most thoroughly and satisfactorily accomplished. No work done upon obstetrics during the past twelve months has been omitted, and the space allotted has been gauged to a nicety to meet the relative importance both of the subject under consideration and the value of the work itself. Thus, a very valuable result has been secured in that the size of the book has been kept within the limits of the ordinary pocket volume.

As an index to the completeness with which the authors have done their work, it may be mentioned that there is a very satisfactory account of the recent investigations of Webster upon Human Placentation, as well as a full consideration of the year's work in the field of local anæsthesia in its relation to obstetrics. W. R. N.

LECONS DE CLINIQUE OBSTETRICALE. (LECTURES IN CLINICAL OBSTETRICS.) Par le DR. QUEIREL, Professor de clinique obstétricale à l'École de médecine de Marseille; Membre correspondant de l'Académie de Médecine. Paris: G. Steinheil, Éditeur, 1902.

THIS series of lectures covers the most important topics in obstetric practice. Obstetric antisepsis forms a proper introduction to the volume. The writer's usage in this important particular is shown to be that of the most intelligent students of the subject. His method of performing craniotomy is by the use of the basiotribe, and in cases where the child is dead and the pelvis considerably contracted, he has succeeded in delivering the patient by symphysiotomy and the use of the basiotribe, thus avoiding abdominal incision.

In a lecture upon pelvic hæmatocele, he describes an interesting case of a nulliparous patient who had amenorrhœa. While having a tooth extracted she was seized with violent pain in the abdomen, with vomiting and partial syncope. She had a slight discharge of bloody fluid afterward. Upon examination the uterus was found the size of the fourth month of gestation. A tumor filled the cul-de-sac, which was soft in consistence and not especially painful. The case was treated by incising the posterior cul-de-sac and turning out a large quantity of clotted blood, douching the cavity with biniodide of mercury solution and packing it with iodoform gauze. The patient perished not long afterward with signs of peritoneal infection.

At autopsy, clotted blood was found throughout the abdomen among the intestines, with beginning formation of pus in the left iliac fossa. The origin of the hemorrhage had been the left Fallopian tube, a pavilion of which was dilated and had evidently contained an ectopic gestation. There was no trace of embryo. This lecture is followed by one upon ectopic gestation, in which a case of abdominal pregnancy is described, successfully treated by abdominal incision with the gradual discharge of the placenta through the abdominal wall.

Abdominal obstruction, rigidity of the cervix, vesicular mole, prolapse of the cord, purpura, epithelioma, and puerperal infection are among the subjects treated in the remainder of the volume.

The author publishes a lecture upon tuberculosis complicating pregnancy, which calls forth an introductory preface from Pinard.

Pinard does not share the view of those who consider tuberculosis an indication for the induction of labor, and he cannot accept the dictum of Peter regarding the tuberculous woman—in youth no marriage; after marriage, no pregnancy; and when a mother, no lactation. Pinard believes that the best results are obtained by treating the tuberculous patient as any other case of tuberculosis would be treated, and by allowing the pregnancy to take care of itself.

The volume concludes with a statistical account of the clinic at Marseilles, covering 827 cases of labor. Among these there were performed the usual operations of obstetrical science, including three symphysiotomies and two entire hysterectomies, one for cancer and one for rupture of the uterus, with infection.

With the exception of a few temperature-charts the book is without illustrations. It is fairly well printed and of convenient size.

E. P. D.

THE PRACTICAL MEDICINE SERIES OF YEAR-BOOKS. II. GENERAL SURGERY. Edited by JOHN B. MURPHY, M.D. Chicago: The Year-Book Publishers, 1901.

THE outward appearance of this book is far from prepossessing, as both binding and paper are of poor quality. One's idea, however, of its worth rapidly changes upon its perusal, as it contains an excellently arranged summary of the best surgical literature of the year. The editorial notes of Murphy, which frequently occur throughout the pages, are very fine. A few articles which might appear to some to be of value are wanting, but it is impossible in such brief space to include all articles, or even to give to the best sufficient space. It would, however, appear to us that excellent judgment had been exercised in the selection and arrangement of the articles for abstraction and comment.

This review of the surgical literature is not as extensive as that found in other publications of a similar nature, but the volume, because of its small size and light weight, will be found very convenient.

The type is remarkably clear considering the very poor paper which is used. Many of the illustrations, however, suffer from the condition of the paper on which they are reproduced.

At the end of the book is found an excellent index which renders ready reference easy.

J. H. G.

ÉTUDES ANATOMIQUES SUR LES GROSSESSES TUBAIRES. (ANATOMICAL STUDIES IN TUBAL GESTATION.) Par le DR. A. COUVELAIRE, Ancien interne des hôpitaux; Chef de clinique obstétricale à la Faculté de Médecine de Paris. Paris: G. Steinheil, Éditeur, 1901.

THIS monograph gives the results obtained in the anatomical study of 35 cases of tubal gestation. The author finds that the ovum lodges in the isthmus of the Fallopian tube much less commonly than in other portions of the tube. In his 35 cases there were but 5 of this sort. In these cases the sac enlarged freely toward the abdomen, pregnancy was interrupted early by apoplexy of the ovum, accompanied by rupture into peritoneal cavity with intraperitoneal hemorrhage.

The ovum lodges far more frequently in the ampulla of the tube. In the author's 35 cases of tubal pregnancy, in 28 the ovum lodged in the ampulla. Of these 28, 8 cases were terminated by apoplexy of the ovum. This happened not by primary rupture of the sac, but simply by the formation of hematosalpinx, with or without hematocele. Primary rupture is much later in ampullar pregnancy than when the ovum is in the isthmus of the tube.

Chapter second of the volume is devoted to the anatomy of the pregnant tube aside from the sac of the embryo. In treating of the reaction which the tube shows to the presence of the embryo, the author finds that the further the pregnancy advances the more pronounced are such modifications. They are also more intense in accordance with the immediate proximity of the sac of the fœtus. These modifications embrace all the histological elements of the Fallopian tube. In studying the anatomy of the ovum in the tube itself the author finds that the chorion of a young tubal embryo is villous throughout its entire extent. Such a young embryo is inserted upon a part only of the wall of the tube. One of its extremities adheres to the Fallopian tube, while the other lies free in the cavity of the tube. The chorion of the tubal embryos not more than three months advanced shows no special development of villi. The placenta which becomes discoid is localized only as a part of the circumference of the embryo. Up to the period of three months the free extremity of the ovum does not contract adherence with the tubal wall. In general, the ovum within the tube undergoes evolution with a discoid placenta. Its relations with the cavity of the tube are identical with those seen between an intra-uterine ovum and the wall of the uterus.

In considering the attachment of the ovum to the wall of the tube the author finds that from the fourth week the border of the zone of insertion of the embryo to the tube, and finally all the tissues, show tubal epithelia. From the fourth week on the ovum in the tube is surrounded by tissue resembling exactly the decidual lining of the uterus. The spaces between the villi are separated from the wall of the tube by a layer of irregular cells whose connective tissue support resembles a network of fibrin. It is also found that large cells are present in the tissues surrounding the endothelium and in the cavities of the uterine sinuses analogous to the cells found in the external capsule of the ovum. There is practically no limit between the external capsule of the ovum and the wall of the sac. In the immediate neighborhood of the capsule external to the ovum the wall of the tube presents a dense infiltration with leukocytes, considered by some to be a reaction limited entirely to

the maternal tissues. The wall of the embryo has an extent in structure differing greatly in proportion to the stage of the pregnancy and to the region of the tube from which the specimen is taken. The extent of the sac is considerably less at the border of the attachment of the sac to the tube than upon the free pole of the tube, and this is true the further away from the point of rupture that one examines the specimen. When a section is made through the mesosalpinx the extent of the sac of the embryo seems increased by the tissues of the mesosalpinx.

The author believes that in the gravid tube from the fourth week to the third month there can be shown a limiting membrane on the side of the cavity of the tube extending to the free extremity of the ovum. This membrane is composed of an alveolar stroma containing abundant cells, with large nuclei. The placenta from the eighth to the twelfth week is developing at the border of the free pole of the ovum and also upon the wall of the tube. The wall of the tube opposite the free pole of the ovum is covered by a lining of mucous membrane. The limiting membrane of the free pole of the ovum does not seem to have the function or position of a reflexa.

In considering the question of the mechanism of the rupture of tubal pregnancy it is found that rupture most often occurs at the parietal zone of attachment of the ovum.

The text of the monograph is supplemented by fifteen clinical observations giving the history of cases forming the basis of this publication. Twenty plates are added as illustrations. The works of those who have written upon the subject are quoted, although there is no considerable bibliography.

This monograph is an interesting contribution upon an interesting and important topic. It deserves a place in the literature of the subject, and should be consulted by those who study tubal gestation. The illustrations are not especially satisfactory, as many of them are inferior in clearness. There are no colored illustrations. E. P. D.

PRINCIPLES OF BACTERIOLOGY: A PRACTICAL MANUAL FOR STUDENTS AND PHYSICIANS. By A. C. ABBOTT, M.D., Professor of Hygiene in the University of Pennsylvania. New (6th) edition, revised and enlarged. In one 12mo. volume of 636 pages, with 111 illustrations, of which 26 are colored. Philadelphia and New York: Lea Brothers & Co.

WITHIN the last few years such advances have been made along the lines of bacteriological research, and so much light has been shed upon the problems of immunity and infection by studies in this direction, that a new edition of this work comes at a most opportune moment. Designed especially for students and those wishing a general practical knowledge of the subject, the book fills a distinct want. The first chapters deal with methods of sterilization, preparation of culture media, and the technique of staining. All the descriptions are clear, and the text is sufficiently illustrated to demonstrate the application of these methods. The technique of animal inoculation is also discussed, and general directions are given for a careful study of bacteria The common bacteria pathogenic for man are described in turn, and attention is paid

to the differential diagnosis of allied micro-organisms. Among the newer features of this edition are summaries of the recent work upon epidemic cerebro-spinal meningitis and dysentery; discussions upon the subject of tuberculosis, together with the group of bacteria in some ways resembling bacillus tuberculosis, and a review of the mechanism of infection and immunity. The portion which deals with the investigation upon dysentery is of special importance, since most of the knowledge acquired upon this subject is of such recent date that descriptions of the bacillary form of dysentery have not yet found their way into general text-books. The chapter upon the subject of tuberculosis is likewise valuable. Some confusion has arisen as to the identification on morphological grounds of bacillus tuberculosis and other acid-resisting bacilli, such as the timothy bacillus, etc. The methods for differentiating these organisms are discussed, and interesting descriptions are given of the pathological lesions produced by them in animals. Reference is also made to the subject of bovine and human tuberculosis, and other additions have been made in the part allotted to the pathogenic streptothrices. Such rapid advances have been made recently in our knowledge of the theories of infection and immunity, and so much work has been done in this branch of scientific medicine, that a complete survey of the subject would, of course, be out of place in a book of this nature. Nevertheless, the history of the development of this side of medicine is well reviewed, and the fundamental conceptions upon which Ehrlich's theory is based are well but concisely given. All confusing details are omitted, and emphasis is especially laid upon the most important points. In fact, the entire work is so written that the essentials are brought out clearly, while the details are sufficiently suppressed to avoid confusion, and the book thus makes a most excellent guide for the student of the subject as well as a useful manual for more advanced workers. W. T. L.

LAMARCK, THE FOUNDER OF EVOLUTION: HIS LIFE AND WORK. With Translations of His Writings on Organic Evolution. By ALPHEUS S. PACKARD, M.D., LL.D., Professor of Zoölogy and Geology in Brown University. New York: Longmans, Green & Co., 1901.

THE life of Lamarck, although not directly devoted to the advancement of medical science, has nevertheless influenced it as the work of Darwin did by its reflex upon all scientific methods of research and modes of thought.

In the preface to this book the author states that "this life is offered with much diffidence, though the pleasure of collecting the materials and of putting them together has been very great." Every page proves the truth of the latter statement, the entire book having evidently been written *con amore*.

Lamarck was born in a little village in Picardy in 1744, and died in Paris in 1829. His long life covered a period of thrilling interest in the history of France and of wonderful progress in the natural sciences. He was intended for the clergy, but being a young man of spirit he cast aside his bands after spending four years in the Jesuit College at Amiens, and entered the French army. He was promoted for bravery, but was obliged to retire from the army shortly afterward because of

physical disability. He returned to Paris, and entered upon the study of medicine, eking out his meagre pension of 400 francs a year by working in a bank.

While pursuing his medical studies he met Rousseau, then living in the neighborhood of Paris, and from him probably received much impetus in his botanical studies. It is of interest, as the author suggests, to find Rousseau foreshadowing natural selection when he says that the weak in Sparta were eliminated in order that the stronger and superior members of the race might survive, and theorizing on the lessons to be derived therefrom in the improvement of the human race.

At the age of twenty-four Lamarck abandoned arms, medicine, and the bank, and commenced his exclusively scientific career. The *Flore Française* was the result of nine years' work, and instantly made him famous. It introduced him to the friendship of Buffon, and that all-powerful man was able to procure him government employment.

The author details the active part taken by Lamarck in having the *Jardin du Roi* changed by the National Assembly to the *Jardin des Plantes*, thereby preserving that most useful establishment from the fury of a mob bent on destroying everything associated with royalty. The organization of this museum is the same now as in 1793, and owes its chief features to Lamarck's genius.

The rest of Lamarck's life was passed in arduous labor in an official capacity in the museum. Successive chapters of the book are devoted to his work in meteorology, geology, invertebrate paleontology, botany, general physiology, and biology, in all of which his views were far in advance of most of those possessed by his contemporaries. The last ten years of his life were spent in blindness and poverty. Although his burial-place has been ascertained by diligent search, the facts revealed concerning his interment were most discreditable. His body was thrown into a trench somewhat apart from the other graves in the Cemetery of Montparnasse, from which the bones of those deposited there were removed every five years. As the author says, they are probably now in the catacombs of Paris, mingled with those of the thousands of unknown or paupers in that great ossuary.

Of course, the main interest in his life attaches to those views which are now embodied in the term "Lamarckism." He on numerous separate occasions promulgated and maintained the great influence possessed by environment in changes and alterations produced in animals, and that species were not immutable but variable. Owing to misinterpretation, to ignorance, and to lack of a proper appreciation, his views, instead of being hailed as those of a great scientific genius, were ridiculed by those who should have regarded them in a very different light. Darwin failed to realize their importance, and publicly decried them. Nevertheless, at the present time, it has become generally recognized that Darwin's theory of natural selection is not adequate solely to account for evolutionary progress, and the influence of environment is being more and more recognized as a necessary factor in conjunction with natural selection.

For one who is championing a cause the author is to be commended for the breadth of view, lack of prejudice, and judicial attitude which he maintains throughout the entire work. We trust that his labor will prove fruitful, and that the great French scientist, whose name and fame have fallen into such ill-deserved neglect, will assume his place as a recognized leader of advanced scientific thought. F. R. P.

OUTLINES OF ANATOMY: A GUIDE TO THE METHODICAL STUDY OF THE HUMAN BODY IN THE DISSECTING-ROOM. By EDMUND W. HOLMES, A.B., M.D. Second edition. Lancaster, Pa.: Press of the New Era Printing Co., 1902.

THIS little book certainly fills a want of every student in the dissecting-room, as it tells him in a simple and interesting way the direction and method which he should pursue in his study of anatomy. It is in no way meant to supplant the text-book or even the quiz compend, but rather to develop in the student a careful and methodical consideration of the subject. So systematic has the author been that the work is divided according to the days to be spent upon each part. To the beginner particularly we can commend this work as a valuable aid to the practical study of anatomy. J. H. G.

RATIONAL HYDROTHERAPY: A MANUAL OF THE PHYSIOLOGICAL AND THERAPEUTIC EFFECTS OF HYDRIATIC PROCEDURES AND THE TECHNIQUE OF THEIR APPLICATION IN THE TREATMENT OF DISEASE. By J. H. KELLOGG, M.D., Member of the British Gynecological Society, etc. With 293 illustrations, 18 in colors. Pp. 1193. Philadelphia: The F. A. Davis Company, 1901.

DR. KELLOGG'S work is an interesting contribution to the literature of hydrotherapy. The entire subject is presented in every phase that may be useful to the practitioner. The dependence of all hydrotherapeutic procedures upon physiological action is judiciously emphasized. The technique of hydrotherapy is described in detail. In the chapter on Hydriatic Prescription-making the author enlarges in a thoroughly practical manner on "procedures to increase and decrease vital resistance, oxidation, metabolism, blood movement, heat production and elimination." Under each caption full instructions with cross-references are furnished, which lead to enlargement of the volume to an unwieldy size. If the reader has the courage and perseverance to follow the numerous pages referred to in the index under each disease he will find guidance to many therapeutic resources when he has become disheartened by failure of drugs and other remedies.

A defect which the author would do well to remedy in future editions is the utter ignoring of his contemporaries in the building up of hydrotherapy in America. He implies that nothing has been done during the last fifteen years by American physicians, and that "we were much nearer the front of this line of progress half a century ago than we are at the present time." The well-informed reader will at once recognize and resent the groundlessness of this charge. Does the author expect his readers to believe that the one hundred and odd American contributors cited in his bibliography have been so barren of results? In defence of his American contemporaries the veracious reviewer would also advert to the author's deplorable unfamiliarity with the literature of his subject. No mention is made of Hiram Corson's earnest plea for cold water and ice in diphtheria and the exanthemata; of Mary Putnam

Jacobi's classical monograph on the wet-pack; of Fredrick Peterson's excellent essay on Hydrotherapy; of S. Solis Cohen's interesting papers; of Carl Seiler's convincing monograph on "The Brand Bath in Typhoid Fever;" nor of Hutchinson's novel views on the skin (peripheral) heart, which term the author uses without even mentioning the originator's name. All these and many other contributions to the advancement of hydrotherapy have appeared in our most prominent journals and have doubtless influenced its practical application enormously. The Montefiore Home for Chronic Invalids of New York and the German Hospital of Philadelphia have *for ten years* had a separate department for hydrotherapy equipped with a douche table devised by an American physician, and which has become part of the equipment of many other institutions. Peterson's essay in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES gave an impetus to systematic hydrotherapy in asylum practice, the marvellous results of which are related in the reports of the St. Elizabeth Hospital at Washington, the Danvers Hospital for Insane, and other institutions. These and many other positive data may be cited to refute the author's unfounded reflections upon American physicians and to disapprove his contention that hydrotherapy has not been furthered by American physicians during the past fifty years. S. B.

EINFÜHRUNG IN DAS STUDIUM DER MALARIAKRANKHEITEN MIT BESONDERER BERÜCKSICHTIGEN DER TECHNIK. EIN LEITFADEN FÜR SCHIFFS- UND COLONIALARTZE. Von REINHOLD RUGE, Marine-Oberstabsarzt I. Kl., Kommandirt zum Institut für Infectionskrankheiten in Berlin. Mit 2 photographischen swie einer lithographischen Tafel, 19 Abbildungen und 27 Fiebercurven im Text. Jena: Verlag von Gustav Fischer, 1901.

ALTHOUGH small, this work is a storehouse of information. Beginning with the geographical distribution and history of the disease, it contains chapters on Epidemiology, Symptomatology, Pathogenesis, Pathological Anatomy, Diagnosis, Prognosis, and Treatment. The history of the malarial fevers is divided into three periods, the first extending from the time of Hippocrates to the year 1640, when the Countess Cinchon, the wife of the Viceroy of Peru, was cured by the use of cinchona bark, and brought the remedy back with her to Europe. The second period comprises the time between 1640 and 1880, when Laveran discovered the malarial parasite. The third period includes the discoveries that have been made since 1880, namely, the observations upon the development of the parasite in human blood by Golge (1885); the conveyance of the parasite to man by the mosquito (Ross, 1898); the work of R. Koch and many others. In this connection it is worthy of note that the ancient Romans were well aware of the more obstinate character of the quartan type of fever as compared with the tertian, and its greater tendency to recurrence—a fact exemplified by the malediction "quartana te teneat!"—"may the quartan fever seize thee!"

Our author does not believe in the independent existence of quotidian fever, for the excellent reason that the only malarial organisms thus far

discovered are those of tertian, quartan, and tropical or, as we call it, æstivo-autumnal fever. A quotidian fever must, therefore, either be a double tertian—*i. e.*, a fever caused by two generations of tertian parasites in the blood—or a triple quartan—*i. e.*, a fever caused by three generations of quartan parasites, in both cases the separate generations sporulating at intervals of twenty-four hours.

The section on treatment, and especially that portion of it relating to prophylaxis, which has acquired a new significance since the mosquito has been proved to be the *fons et origo mali*, is not inferior to the other portions of the work. This is equivalent to saying that it is concise and complete. Quinine is the remedy *par excellence*, with one exception, namely, in cases of "blackwater fever." When hæmoglobin appears in the urine, or when there is a history of hæmoglobinuria in previous attacks, methylene blue, the only remedy, besides quinine, which has a direct effect upon the malarial parasite, should be employed.

The only criticism we have to make of this excellent treatise is the absence of reference to the American savants who have contributed so extensively to the literature of malaria, and whose names should be, in Europe as they are here, "familiar as household words." This is in marked contrast to our custom, which is rather to exaggerate the importance of the work of foreigners and belittle that of our own countrymen. In this attribute, which partakes of false modesty rather than of generosity, we resemble the Italians, who, because of it, have just received a well-merited rebuke from Cesare Lombroso.[1] F. P. H.

STUDIES IN PHYSIOLOGICAL CHEMISTRY. Edited by R. H. CHITTENDEN, Ph.D. Sheffield Scientific School, Yale University, 1901.

THAT ancient and revered institution known as Yale University has just celebrated its bi centennial. As a part of the celebration of this anniversary the University has published a series of volumes by a member of the professors and instructors of the faculty that serves as an indication of the extent and character of the work done and the progress that is being made, and of the wonderful growth of the University.

The volume before us is a thick 8vo. of 424 pages, giving reprints of some of the work recently done in the University under the able direction of Dr. Chittenden, Director of the Sheffield Scientific School, and his collaborators. It is prefaced by a bibliography of work done in the field of physiological chemistry at Yale since the establishment of the Sheffield School, in 1875, up to the present time, a list embracing 104 titles of papers published both in this country and in Germany.

The book contains reprints of twenty four previously published articles, and is the work of Drs. Chittenden, Mendel, McDermott, Van Name, Giess, Jackson, Richards, Henderson, Brown (E. B.), and Alice H. Albro, many of the papers being done in conjunction.

[1] "The Anti-Italianism of the Italians," Living Age, July 13, 1901.

The papers contained in the book are mainly technical, but there are some that will be of interest and value to the general practitioner.

Dr. Lafayette B. Mendel contributes an article on "The Chemical Composition and Nutritive Value of Some Edible American Fungi." The article contains a mass of detailed work, including many analyses and a number of artificial digestion experiments. Fungi contain from 75 to 90 per cent. (and sometimes even more) of water, and, as the greater part of the solid matter is carbohydrate, we can hardly speak of them as "Vegetable Beefsteak," as some enthusiasts term them. The total amount of proteid in them, as figured by modern and more correct methods, is strikingly small, not above 5 per cent. figured as "nitrogen" and on the dried substance. The carbohydrate is relatively high, but very little is known as regards the nature and digestibility of this constituent. The author sums up his work by saying: "The fungi thus form no exception to the ordinary classes of fresh vegetable foods; indeed, they take a decidedly inferior rank in comparison with many. . . . As dietetic accessories the edible fungi may play an important part; but investigation has demonstrated that they cannot be ranked with the essential foods."

Dr. Ernest W. Brown has a short but interesting contribution on "Cetraria Icelandica" (Iceland moss). He gives an analysis of the dried substance. The principal constituent is "lichinin," and from his experiments on living animals he finds it an excellent food and easily assimilated. The amount of the proteid material is extremely small, and the main bulk of the material (exclusive of the water present) is made up of the soluble carbohydrate, the so-called "lichin-starch."

An extremely valuable paper, and, perhaps, the most important in the volume, is one entitled, "A Further Study of the Influence of Alcohol and Alcoholic Drinks on Digestion, with Special Reference to Secretion." This is contributed by Dr. Chittenden in conjunction with Drs. Mendel and Jackson. The article, however, is too lengthy and too technical to admit of abstraction here. E. A. C.

FORMULAIRE AIDE-MEMOIRE DE LA FACULTE DE MEDECINE. By DR. FERNAND ROUX. Paris, 1902.

DR. FERNAND ROUX has prepared a very valuable little book for the use, apparently, of hospital residents, who form, as is well known, a distinguished class in Paris. It consists of an alphabetical list of most of the known diseases and diseased conditions, with a synopsis under each of the treatment according to various authorities, including numerous formulæ. Naturally, as it is merely a collection of citations, there is nothing original, and, therefore, nothing to criticise except the method. This seems to be very good. The descriptions are very clear, and the formulæ sufficiently diversified. As an appendix there are some suggestions regarding the writing of prescriptions, and a brief note on some of the newer remedies. The latter does not seem to be as complete as one could wish. The book is compact in spite of its 538 pages, is substantially bound, and very clearly printed. J. S.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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On the Surgical Treatment of Ascites.—The purely medical treatment of ascites is, on the whole, so unsatisfactory that it is well for physicians to be familiar with the results of surgical procedures up to date.

MONTGOMERY (*Medical Chronicle*, April, 1902, p. 15) gives a brief historical sketch of the steps which led up to the adoption of surgical measures for the treatment of ascites. Hale White, in 1893, showed that the average duration of life of cirrhosis patients, from the necessary first tapping until death occurred, was sixty-three days; while in cases which were not subjected to paracentesis the average length of life from the beginning of enlargement of the abdomen until death was sixty days. In other words, paracentesis had practically no effect in prolonging life. He believed that when ascites is survived for a much longer time it is due to a coexistent chronic peritonitis and perihepatitis, with the consequent establishment of a collateral circulation between the portal and systemic circulation.

Talma, of Utrecht, was the first to suggest that such an artificial anastomosis be brought about by operation. Following this suggestion the first operation was performed by Van der Meule in 1889, the patient dying of shock. The third case was operated on by Lens in 1892. This is the first reported case. The patient recovered from the operation, but his symptoms were not relieved.

Drummond and Morrison appear to have independently conceived the same idea as Talma. In 1894 and 1895 Morrison operated on the first two cases in England. The first case was not benefited, and died nineteen months later. The ascites in the second case entirely disappeared, but the patient died two years later from a hernia operation. At autopsy it was shown that extensive omental adhesions had resulted from the operation.

The operations for the relief of ascites all have for their object the establishment of a collateral circulation between the portal and general venous systems. This is produced through the establishment of peritoneal adhesions. To effect this result the omentum is generally utilized in one or other of the following ways: (1) It may be sutured to the parietal peritoneum alone. (2) It may be fixed under the skin of the abdominal wall. (3) It may be sutured between the liver and diaphragm. The parietal peritoneum may be separated from the muscles around the abdominal incision and the omentum grafted into the prepared space. At the time of the operation it is customary to scrub the surface of the liver, diaphragm, and spleen to promote their subsequent adhesion.

Montgomery reports two cases on which he himself has operated. In one case the ascites apparently was cured, as it had entirely disappeared six months after operation. The other case was only slightly relieved.

Since the analysis by Packard and Le Conte of 22 cases collected from the literature, Montgomery has found records of 16 others, including his own, making a total of 38 cases. The result in these cases was as follows: 10 deaths, or 26.3 per cent.; 9 unimproved, or 23.6 per cent.; 4 improved, or 10.5 per cent.; 15 recovered, or 39.4 per cent.

On Adrenalin Glycosuria and Other Forms of Glycosuria Due to the Action of Reducing Substances and Other Poisons on the Cells of the Pancreas.—Some months ago the experiments of F. BLUM on adrenal diabetes were noted in this department. Blum found that the subcutaneous injection of an aqueous extract of adrenal glands produced glycosuria in twenty-two out of twenty-five animals experimented upon. Recently Herter (*American Medicine*, May 10, 1902, p. 771) has carried out some instructive experiments showing the effect that adrenalin chloride and solutions of other reducing substances have when painted over the surface of the pancreas.

Commercial aqueous solution of adrenalin chloride 1 : 1000 was used in the research. Herter first showed that the subcutaneous, intravenous, and intraperitoneal injections of adrenalin chloride into dogs were almost invariably followed by glycosuria. Peritoneal injection, other conditions being equal, produced the most marked glycosuria, 10 per cent. or more of sugar not being uncommon. The glycosuria lasts usually somewhat less than twenty-four hours. It was important to find whether the more marked results following intraperitoneal injection of adrenalin chloride were due to the opportunity for more direct action of this substance on the pancreatic gland. Accordingly the pancreas was exposed in a number of normal dogs and adrenalin applied directly to the presenting surface by means of a brush. It was demonstrated that marked glycosuria follows the application of small quantities of adrenalin to the pancreas, quantities which, when applied locally to other parts of the body, either give rise to no excretion of sugar or to a trivial glycosuria.

The next problem was to determine how the local application of adrenalin to the pancreas produced glycosuria. It was first shown by experiment that it was not due to vascular disturbance. Adrenalin chloride is known to be a very powerful reducing substance, and it was decided to ascertain the effect that other reducing substances had when applied directly to the pan-

creas. The thought suggested itself that the use of such reducing substances might interfere with oxidation within the pancreatic cells. Local applications of potassium cyanide were first tried and constantly produced a glycosuria. Similar results were obtained with sulphurous acid, ammonium sulphide, sulphuretted hydrogen, illuminating gas, carbon monoxide and several other reducing substances. A number of similar experiments were carried out with substances which do not reduce, such as sodium chloride, sodium hydroxide, ferric chloride, hydrochloric acid, bromine water, etc., without the production of glycosuria in any instance.

Herter concludes from these experiments that there is a close relationship between the reducing power of a substance and the capacity of this substance to induce glycosuria when applied directly to the pancreas. He believes that the amount of oxygen which these substances remove from the cells of the pancreas is sufficient to cause a disturbance of the function of the gland.

Histological examination of the gland tissue at the height of the glycosuria fails to show any recognizable lesion. Herter does not believe that the glycosuria is due to disturbance of the function of the islands of Langerhans.

It is pointed out that these experiments are of clinical importance, in that they probably throw considerable light on several varieties of human glycosuria and diabetic cases. Herter suggests that the glycosurias following conditions of asphyxia, as after epileptiform seizures, may be due to interference with oxidations in the pancreas. So also with the glycosuria following carbon monoxide poisoning.

Contribution to the Study of the Myospasms: Myokymia, Myoclonus Multiplex, Myotonia Acquisita, Intention Spasm.—WALTON (*Journal of Nervous and Mental Disease*, July, 1902, p. 402) draws attention to the fact that there is much confusion in certain text-books, and often on the part of writers, in the classification of the various anomalies of the neuromuscular mechanism which gives rise to involuntary spasmodic movements. The writer endeavors by means of the reports of illustrative cases to clear up certain misconceptions which have led to error in the classification of these interesting cases. His conclusions are as follows:

(1) The term *myoclonia*, as a collective designation for the unrelated disorders, should be discontinued.

(2) The term *myokymia* should be limited to cases showing, without hereditary or congenital history, widespread muscular quivering, without atrophy or other indication of progressive degeneration of the nervous system, without constitutional symptoms, and without sign of present infectious or other acute disease. Cases should not be excluded, however, on account of preceding or introductory symptoms pointing to disease of the lower neurons, if such disease has either disappeared or come to a standstill.

(3) The term *myoclonus fibrillaris multiplex* should not be applied to such cases, since it suggests a relationship between myokymia and the paramyoclonus multiplex of Friedreich, with which it has nothing in common.

(4) *Myoclonus multiplex* (the prefix para seems superfluous) should be used to designate bilateral clonic spasms involving whole muscles or groups of muscles, generally those attached partly or entirely to the trunk.

(5) The term *myotonia acquisita* should be limited to non-heredity and non-congenital cases, in which otherwise healthy individuals present the typical motor disorder or the typical reactions of Thomsen's disease. This term should not include the rigidity accompanying marked intestinal disorder or pronounced psychopathic states, even though the rigidity in the latter conditions may be increased by involuntary movement.

(6) The tendency to spasm on attempted voluntary movement, unless accompanied by the typical motor disorder or the typical reactions of Thomsen's disease, should be classed as *intention spasm*.

(7) The term *myospasm*, clonic or tonic, may be used instead of myoclonia and myotonia when it is desirable to include under one head the various forms of involuntary muscular contraction without known organic basis.

Concerning the Benzoyl Esters of the Urine in Diabetes Mellitus, and the Clinical Significance of an Excess of Glycuronic Acid.—EDSALL (*University of Pennsylvania Medical Bulletin*, April, 1902, p. 34) points out that Mayer has shown that glycuronic acid is a normal constituent of the urine, and that it can be easily recognized by the orcin test, if the glycuronic acid compounds be previously split up by heating with dilute sulphuric acid. Mayer believes that glycuronic acid is one of the steps in the normal oxidation of sugar, and, owing to the close chemical relation between glycuronic acid and glucose and the frequency with which he found glycuronic acid in cases where there was suboxidation, he believes that he has demonstrated definitely that diabetes is due to suboxidation. The clinical importance of the presence of glycuronic acid in the urine is that it is probably more frequently mistaken for glucose than any other carbohydrate. Glucose and glycuronic acid are then both found normally in the urine, and under certain conditions both are increased. According to Mayer, it would appear that glycuronic acid had much the same significance that glucose has.

Rosin and v. Alfthan demonstrated that the benzoyl esters are increased in diabetes mellitus. Mayer believed that this increase was due to an increase of the glycuronic acid, and not to an increase of the unfermentable carbohydrates, as Rosin and v. Alfthan believed. Edsall agrees with the latter view, and found in three cases of diabetes in which glycuronic acid was absent that the benzoyl esters amounted respectively to 12.56, 13.43, and 13.88 grammes. The normal is less than 5 and not more than 2 or 3 grammes.

The importance of an increased elimination of glycuronic acid and of the benzoyl esters in diabetes mellitus is, considering that both belong to the carbohydrate group, that it demonstrates that the metabolic disturbance in diabetes does not involve the dextrose alone, but that there is in this disease an actual disturbance of the whole carbohydrate metabolism. Edsall has shown that the source of the benzoyl esters is chiefly the proteid elements of the food, and that the amount varies directly with the variations of the diet.

Eosinophilia Associated with Hydatid Disease.—SELIGMANN and DUDGEON (*Lancet*, June 21, 1902, p. 1764) report an instance of marked eosinophilia in association with hydatid disease of the liver in a young woman, twenty-two years of age. The disease was probably contracted as a result of fondling a pet dog which constantly licked her hands.

The first symptoms of the disease began in December, 1900, with pain in the right side. In December, 1901, she was admitted to St. Thomas' Hospital with marked enlargement of the right lobe of the liver and a friction rub in the right axillary region, which led Dr. Hector Mackenzie to suspect hydatid disease. On December 27th the blood examination gave the following results: Red cells, 6,290,625; leucocytes, 17,000; hæmoglobin, 70 per cent. A differential count of 500 leucocytes was as follows: Polymorphonuclear neutrophiles, 22 per cent.; eosinophiles, 57 per cent.; lymphocytes, 20 per cent., and basophiles, 1 per cent.

On January 10, 1902, the patient was operated on, ten ounces of clear fluid containing hooklets being removed. After the operation the percentage of eosinophiles gradually returned to normal.

The writers state that the percentage of eosinophiles in this case is only exceeded by Brown's 68 per cent. of eosinophiles in a case of trichinosis. It is approached by Billings' findings of 53.6, 38.2, and 33.9 per cent. respectively in three cases of bronchial asthma.

SURGERY.

UNDER THE CHARGE OF

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The Treatment of a Case of Scirrhus Recurrent Five Years in a Patient Aged Ninety-three Years; Improvement.—PETERS (*British Medical Journal*, March 1, 1902) reports the case of a woman, aged ninety-three years, who had a recurrent slow-growing spheroidal-celled carcinoma. The original growth had been removed thirteen years ago, but eight years later recurrence took place, and at the date of the commencement of the Röntgen treatment the growth, which was adherent to a rib, measured 3 by 2¾ inches. It had a well-raised margin, covered by thin, red epidermis, while the centre was an irregular ulcer. There was also some ulceration of the raised margin, with large tender glands at the edge of the lesser pectoral muscle. The exposures were made two or three times a week for from ten to twenty minutes. The distance of the tube from the skin varied from two to four inches. Unfortunately the patient died from pneumonia after ten exposures had been given. Improvement was noticeable at the end of a week's treatment, and on examination after the last treatment it was found that the character of the growth had completely changed. It then measured 1 by ¾ inches, the margin was only slightly raised, and the central ulcer had markedly decreased in size.

Suture of Heart Wounds.—(The Oration on Surgery at the 1902 Meeting of the American Medical Association.) SHERMAN (*Journal of the American Medical Association*, June 14, 1902) states that thirty-four operations have been recorded in the six years which have elapsed since the date of the first operation.

As regards the manner of wounding, all these cases, except two, were due to punctured or incised wounds, the two exceptions being bullet wounds. The particular injury to the heart was inflicted on the ventricles thirty-two times, the left ventricle being implicated seventeen times and the right thirteen times. In two cases only were auricles opened, once the right and once the left, and there are three cases in which my information is incomplete. In most of the cases where details are given the pleura is reported wounded, and usually there was a hæmothorax, the collection representing, in large part, the overflow from the pericardium.

The practical questions which usually come to the mind of a surgeon in planning an operation to meet these conditions relate to the method of exposure of the heart, the detail of the treatment of its particular condition, and the method of closing and dressing the wound of the operation.

In the cases reported the heart was reached variously, depending on the location of the original wound in the skin and the choice of the operator, but either a flap or all of the tissues of the thoracic wall was turned up, or a resection of two or more ribs was practised. The particular detail is of no great moment, provided that the heart is properly exposed. The special method of treating the heart wound is of interest, for it involves the choice of suture material for a novel situation, the time at which the suture is introduced and tied with reference to the heart-beat, and the depth of the stitch in the heart muscle. In our cases three operators are recorded as having used catgut, Fontan, of Paris, Marion, and Launay; in all other instances where the suture material was specified silk was used, and in most cases the sutures were interrupted, though in a small number a continuous suture was practised. It is of interest to note that these operators particularly avoided including the endocardium in the suture. One of them definitely reports introducing and tying the suture during diastole.

As regards the closure of the wounds—the tissues involved in the flaps or incisions were, of course, replaced; in seven instances drainage of the pericardium and pleura was practised, and in four the pleura alone was drained. The other cases are said plainly to have been closed without drainage, or nothing is said of the matter at all.

Now, of the total number of these cases, five died on the operating-table of hemorrhage, and ten died very soon afterward of the effects of hemorrhage or the shock of the operation; so that nearly half of the cases that survived the injury long enough to be subjected to operation died during or very shortly after that operation. The other group, nineteen in number, had various results, but thirteen of them recovered, and only six died. I think it is fair to stop a moment and consider these facts. Surely, the fifteen who died of hemorrhage or shock with operation would probably have died of hemorrhage exactly the same without operation. No fatal traumatism is inflicted by exposing the heart; and stopping the hemorrhage from an incised or punctured wound in the ventricles is a simple matter when the heart

is once exposed; indeed, pressure with the finger or a tampon will stop it temporarily. At all events, the operation and suture did not add materially to the amount of blood lost, and so cannot be counted as having hastened the death from hemorrhage, and the average amount of shock I cannot estimate. But I believe that it is fair to say that these patients had, from the first, practically no chance to recover, and that, if this had been known, the operations need not have been done; but I say this with full appreciation of the fact that the inevitable fatality could not have been definitely predicated in any case. Of the other nineteen, the comment is, plainly, that they had a chance to recover. In each of them the suture of the heart was a successful procedure; in not one instance was the fatal outcome due to a secondary hemorrhage. The six who died succumbed to the common matter of an infection sequent to wound and operation. Of the thirteen who recovered four did so in spite of a concurrent infection. But the point is that of these thirty-four cases, fifteen had really but very slight expectation of benefit from the operation, and died probably neither in spite of it nor because of it—nineteen had expectation of recovery from operation, and in thirteen that expectation was realized.

Analysis of Ninety-six Operations for the Relief of Tuberculosis of the Testicle.—HORWITZ (*Journal of the American Medical Association*, June 21, 1902), after a detailed review of this interesting condition, states in conclusion:

1. A primary tubercular infection of either the epididymis or testicle may occur, the former being by far the more common.

2. A primary infection of the epididymis, secondarily that of the testicle, is more common than the descending one.

3. Primary involvement of either the epididymis or testicle usually takes place through the circulation, the soil being predisposed to the location of the tubercle bacillus either by a slight traumatism or by some infective condition which has given rise to inflammation of the organ, most commonly an attack of gonorrhœa.

4. Secondary tubercular involvement of the epididymis or testicle sometimes follows a primary focus of the disease in other portions of the body, more commonly in those organs that are in direct anatomical connection with the sexual glands, such as the seminal vesicles, prostate, urethra, bladder, ureter, or kidney.

5. The invasion of the testicle may be rapid, associated with acute inflammatory symptoms, an abscess soon developing; or the onset may be slow, the symptoms simulating those of either chronic syphilitic orchitis, or malignant disease of the organ.

6. The tuberculin test should always be employed in doubtful cases where only one focus of the disease is known to exist.

7. In doubtful cases associated with hydrocele, the fluid should be examined for the tubercle bacilli and inoculating experiments made.

8. The injections of either emulsions of iodoform or of sulphate of zinc into the diseased part are not to be recommended.

9. In all cases of encapsulated caseous nodules quiescent in the epididymis, epididymectomy should be performed.

10. Epididymectomy, together with resection of the vas deferens, is not attended by either atrophy of the testicle or sexual weakness.

11. The drainage of tubercular abscesses followed by the use of the curette is only to be employed where radical treatment is not permissible, as it is attended with more or less danger, and is generally unsatisfactory in its results.

12. In instances where the epididymis alone is involved, a resection of the diseased structure is all that is required; whether a partial or complete resection of the vas deferens is to be undertaken is still undetermined.

13. Double orchidectomy should be performed when both glands are diseased, provided there is not extensive co-existing tubercular infection of other organs.

14. Whether infected seminal vesicles should always be removed at the time that the epididymis or testicle is resected is a question open for discussion. From the fact that in a large majority of cases the removal of the primary seat of the disease is followed by a subsidence of the tubercular involvement of the vesicles, it is deemed wiser, as a rule, to wait and remove the vesicles later, if necessary.

15. Hygienic and climatic influences play as important parts after operations in fortifying the constitution against further invasion as they do in other tubercular conditions.

16. The anti-tubercular remedies are of great value in controlling the disease, and should always be employed in conjunction with whatever surgical procedure may be deemed necessary.

External Œsophagotomy for Cicatricial Strictures of the Œsophagus.—LAMBOTTE (*Journ. de Chir. et Annales de la Soc. Belge de Chir.*, March–April, 1902) states that the treatment of these strictures is full of difficulty, and that complete recovery, when possible, can only be obtained by prolonged treatment. The situation of the thoracic œsophagus, which is the usual site of these strictures, makes their approach a matter of danger and difficulty. The resection of the stenosed portion has been proven impracticable by actual experience. The method of treatment by internal œsophagotomy presents such dangers as to preclude its use. Dilatation is in some cases the only resource at the surgeon's disposal, but the improvement under this method is usually slow, and much time of necessity must elapse before a definite result is obtained, and even then it is necessary to pass a sound at intervals in order to prevent further contractions. External œsophagotomy has proven in the author's hands to be the best method of treatment of this obstinate and distressing affection, and, in conclusion, he reports two successful cases with the technique in detail.

Muscular Contraction in the Secondary Stage of Syphilis—BEGOT (*Gaz. Hebdomadaire de Med. et de Chir.*, June 5, 1902) states that the muscular contraction occurring during the secondary stage of syphilis usually affects the biceps muscle. They are sudden in onset, and are characterized by an inability to fully extend or flex the affected arm. The course of the lesion is variable; it may last several days or several months. It is not the result of any local lesion in the muscle, but is purely a nervous phenomenon. The best treatment consists in injections into the affected muscle of distilled water.

THERAPEUTICS.

UNDER THE CHARGE OF

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A Substitute for Iodoform.—DR. A. FROMMER has employed bone charcoal in a series of cases including bone and joint tuberculosis and suppurating lymph nodes, as it has been asserted that the specific action of iodoform on tuberculous tissue was not peculiar to that drug, but that like properties were possessed by other substances, among them bone charcoal. A 10 per cent. sterilized glycerin emulsion was injected. The results obtained afforded no encouragement. The injection in pure bone and joint cases was always followed by a painful swelling, with rise of temperature, which lasted several weeks and sometimes resulted in acute abscess formation. The only evidence of value was found in the after-treatment of atypical resected joints and of excised lymph nodes, but even here the granulations formed were pale and flabby. Another objection is the subsequent pigmentation of the skin. The author concludes that there is nothing to warrant the substitution of bone charcoal for iodoform. The toxic effects of the latter, he claims, are due to use of an emulsion which has not been sterilized or else exposed to the action of sunlight. In 1000 parenchymatous injections only a slight infection of the needle track had been noted in a few cases. The presence of free iodine in the urine for two or three weeks the author cites as proof that the iodoform is broken up and distributed through the system, thus having a general as well as a local effect. This fact can also be taken as grounds against the substitution of iodoform by a chemically indifferent substance, such as charcoal.—*Deutsche medicinische Wochenschrift*, 1902, No. 12, p. 203.

Pharmacology of Iron.—DR. J. AULDE believes that attention to certain combinations of iron with other drugs will much enhance its value as a tonic. The indiscriminate use of iron as a tonic without attention to the particular indication results in many unsatisfactory effects which are charged to the account of iron. As a hematinic it must be recognized as a superior remedy, but it must be given in small doses, so that the degree of irritation shall not exceed that of gentle stimulation. When impaired nutrition is the dominant factor, it may be used in nearly all cases. The addition of nuclein from animal sources is recommended in acute as well as in chronic cases of dyscrasia, thus favoring cell activity. This is especially valuable in cases of catarrh of the mucous membrane, and stress is also laid upon the use of the arsenate in these cases on account of the influence which arsenic has upon

systemic metabolism. Attention is called to the exceptional value of iron in combination with zinc as a protoplasmic stimulant.—*St. Louis Courier of Medicine*, 1902, vol. xxvi. p. 224.

Poisoning from the Application of Carbolic Acid to the Unbroken Skin.—Dr. J. W. Wainwright reports the case of a patient who had previously used a 2 per cent. carbolic acid solution for pruritus, and who changed the strength of the solution to 4 per cent. to get a more satisfactory anæsthetic effect. After a bath at midnight the solution was freely applied over the abdomen, pubes, thighs, and lumbar region. Seven hours later intense pain was felt, apparently in the bladder. There were frequent chills, profuse cold perspiration, vertigo, vomiting, and a desire to evacuate the bowels. The eyes were prominent and fixed. Vertigo increased, with an appreciable paralysis of the lower extremities and complete cessation of the functions of the stomach, bowels, and kidneys, no urine being excreted for eighteen hours. Four hours after the beginning of the attack the patient became drowsy and was awakened from time to time by recurrent lancinating pains. Enemata of hot water were retained. The urinary secretion gradually returned after a hot bath and compresses. Examination of the urine showed it to be smoky and to contain carbolic acid and abundant albumin. The appearance of the urine led to the discovery that the patient had used a carbolic acid solution as described. There was great depression for three days, with evidences of highly inflamed kidneys. In ten days the patient was entirely well. The carbolic acid was evidently absorbed more readily because of the ideal conditions following the hot bath.—*Boston Medical and Surgical Journal*, 1902, vol. cxlvi. p. 361.

Narcotics in India.—Dr. Kailas Chunder Bose reports a series of such cases, most of which had a fatal termination. The sensitive, high-strung Hindu, far more excitable than Europeans psychically, naturally falls an easy prey to the various narcotics. Cannabis indica, as is well known, produces in the marvellously refined nervous economy of the Indian ecstatic dreams of a type which cannot be awakened in the clumsy mechanism of the Occidental. Cocaine, which has but recently been introduced in India, has taken very largely the place of the less harmful gapja or alcohol or bhang. Unlike these, it requires but a simple preparation. It is generally taken in the form of powder sprinkled on a paste of slaked lime, which is buttered on a betel leaf. The mass is rolled up and chewed for about fifteen minutes. The first symptom of the so-called hilarity is a heaviness of the head. Then quickly follow a wild throbbing of the arteries of the neck and palpitation of the heart. The pulse never exceeds 110. The inebriate wishes to be left alone; he will not speak lest saliva escape from the mouth. The ears become hot, the cheeks pale, the nose pinched and cold. The height of intoxication is marked by coldness of the finger-tips and dilatation of the pupils. This stage lasts from thirty to forty-five minutes, when the victim longs for a fresh dose. The teeth and tongue of old habitues turn absolutely black. The craving for an increased dose is pronounced. In one case it was so marked as to cause a jump from one to twenty grains in a month. The worst sequelæ are very obstinate forms of

diarrhœa and dyspepsia. Of the mental derangements, hallucinations, and delusions causing dejection and fear are common. A more miserable object than a confirmed Hindu cocaine-eater cannot be pictured. The drug is altogether more disastrous in its effects than is opium or any other narcotic used in India. To quote the words of a victim, "To eat cocaine is to court misery." It has none of the joys of opium, and is infinitely more destructive.—*British Medical Journal*, 1902, No. 2156, p. 1020.

A New Arsenic Preparation (Atoxyl); its Use in Skin Diseases.—Dr. W. Schild has been studying this drug before attempting its use in practice. Atoxyl is a white, odorless powder containing about 37 per cent. of arsenic and soluble in five parts of warm water. In its effect upon animals the toxicity was found to be only one-fortieth that of the quantity of arsenic which it contains. Hence it affords, according to the investigator, a means of introducing into the system a larger quantity of arsenic than can be introduced safely by other means. Small doses by mouth readily produce gastric disturbance. Hence the author decided to administer the drug only by hypodermic injection. Beginning with two-thirds of a drop of a 20 per cent. solution, the dose is gradually increased until it reaches five times this quantity, at which point it remains during the remainder of the period of treatment. Temporary discontinuance of treatment is occasionally necessitated by the appearance of chills, dizziness, and headache. Favorable results were obtained in cases of psoriasis, lichen ruber, and other chronic dermatoses. While atoxyl will not effect a complete cure of psoriasis unaided by external treatment, neither will other forms of arsenic. It, however, materially aids and quickens the action of external treatment. In fourteen cases of lichen ruber treated exclusively by atoxyl injections complete cures were obtained without a single exception. The average duration of the period of treatment in these cases was fifty days.—*Berliner klinische Wochenschrift*, 1902, No. 13, p. 495.

Influence of Alcohol on Metabolism in Fever.—Dr. A. Ott has found by careful analysis, as have others, that alcohol in fever prevents proteid dissolution to about the same degree as an isodynamic quantity of carbohydrates. The conclusions to be drawn are that, though alcohol should not be thus employed as food, no contraindications exist as to its use in fever in moderation, and especially in the chronic fevers of pulmonary diseases its general stimulation is but desirable.—*Archiv für experimentelle Pathologie und Pharmakologie*, 1902, vol. xlvii. p. 267.

Blood Poisons.—Dr. A. Keil has studied one of the phases of this subject. The various drugs which have a deleterious effect upon the blood may be conveniently divided into six classes. The first comprises the agglutinins, which act by changing the physical conditions of the red blood cells so that they adhere to each other and thus occlude the vessels; ricin and abrin are the two best-known examples. Hæmolysins form the second group; these dissolve out the hæmoglobin which deposits in the liver and kidneys, while the stromata of the cells form multiple capillary emboli; arsenuretted hydrogen, phallin, helvellic and quillajic acids, senegin, sapotoxin, and

solanin belong to this group. Potassium chlorate, pyrogallic acid, and chrysarobin constitute the third class, which is characterized by the formation of methemoglobin. Hydrocyanic acid and carbon monoxide belong to the fourth, in which special hæmoglobin compounds are formed. Fifteen metals are capable of entering into union with hæmoglobin, thus giving rise to the fifth group, while the sixth is made up of substances such as phenylhydrazin and aniline, which are capable of producing partial necrosis of the erythrocytes. The author has studied the effects of the metals on the blood as revealed by the microscope. Granular degeneration of the red cells was present to a marked degree in all animals, and in those with nucleated red cells it could be seen that the process was really a disintegration of the nucleus within the cell or after extrusion. In mammals, however, normoblasts seemed to play no part in the process. It is probable that the granules represent metallic hæmoglobin compounds.—*Archives International de Pharmacodynamique et de Thérapie*, 1902, vol. x. p. 121.

Poisoning by Mussels.—Dr. J. Thesen has been studying one of the phases of this subject. The symptoms following the ingestion of poisonous mollusks may assume one of three types, viz.: (1) The erythematous and least dangerous form, a rapidly developing exanthem of a very transient nature appears, and with it there is general malaise and headache. (2) The symptoms may be chiefly intestinal, and there may appear a more or less severe gastro-enteritis shortly after partaking of the mussel, or this comes on after a period of incubation and is accompanied by fever. (3) The most severe or paralytic type, characterized by acute, peripheral paralysis and resembling curare poisoning. The author has studied this last form. In the cases observed by him either death set in so rapidly through paralysis of respiration that no real dyspnœa was present or else the symptoms were very transitory. All the muscles of the body may be involved without any definite order, and psychical excitement and muscular ataxia may be pronounced. The mode of death, with intact consciousness, is also peculiar. Nausea, with vomiting, generally occurs, but the latter symptom is not forcible or characteristic. Constipation generally exists. With so rapid a course all therapy is worthless, but if seen in time the stomach must be washed out, the patient ordered to be as quiet as possible, and heart and respiration stimulated in the usual way, if flagging. Those patients who come to autopsy have shown some swelling of the spleen and lymph nodes of the intestines with slight gastro-enteric irritation.

To study the toxicity of the mussels they were removed from their shells, placed in cold 96 per cent. alcohol for twenty-four hours, dried on a water-bath comminuted by triturating with sand, and then extracted by boiling with alcohol, acidified with hydrochloric acid. The alcoholic solution was then evaporated and the residue dissolved in water, and this extract employed for subcutaneous injections into animals. Great variations were found in the degree of toxicity, for while the extract prepared from mussels from a neighborhood where fatal accidents had occurred killed mice, rats, and rabbits in a few minutes, those from other places, especially if collected later in the year, were much less poisonous, and even in some instances harmless. Where death occurred the symptoms were in general the same, the trunk

and the extremities quickly became paralytic, respirations were labored, and immediately before death clonic convulsions set in. If the doses were smaller the animals appeared normal for the first few minutes; soon, however, a peculiar motor unrest was evident; then the gait became unsteady, the animal fell, and the entire body lost tone. The extremities could still be moved volitionally, but with pronounced ataxia. There never was diarrhœa. In cats the pupils may become dilated, and the heart generally continues beating after the dyspnœa has led to respiratory paralysis. Examination of the different organs of the mussels showed that the poison is distributed over their entire body, but that the liver contains most of it. In a few cases the water in which the animals lived also proved toxic to mice. The chief factor which renders the mussels poisonous seems to be contamination of the sea in which they live. Either the impurities render the molluscus sick, or such impurities are stored up in them and are themselves the toxic agents. Sections made of the animals, however, failed to reveal any pathological changes in their organs, and the second supposition seems more probable, since the author found that the mussels would readily take up curare, strychnine, and the poisonous extract if added to their water without themselves becoming sick. It thus seems that they have a definite function in the stagnating water of harbors, and that by rendering harmful substances innocuous they purify it. The poison is readilysoluble in water and alcohol, decomposes with alkalies and by bacterial action, but it could not be isolated in pure form.—*Archiv für experimentelle Pathologie und Pharmakologie*, 1902, vol. xlvii., Nos. 5 and 6, p. 311.

GYNECOLOGY.

UNDER THE CHARGE OF

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Cancer of the Uterus.—KRÖMER (*Centralblatt für Gynäkologie*, No. 21, 1902) reports 102 cases of vaginal and 10 of abdominal hysterectomy, with a primary mortality in the former of 3.9 per cent., in the latter of 30 per cent.; 35 per cent. of the cases of cancer of the uterus examined by him were suitable for operation. The proportion of cures in the vaginal cases was 12.8 per cent.; after the abdominal four out of seven who survived were still free from recurrence. The writer rejects the latter method on account of the higher mortality and the impossibility of removing all the lymph nodes and vessels, as in the radical operation for cancer of the breast. So long as the difficulty of diagnosing infection of the glands exists, and the remote results are still uncertain, he will continue to perform radical vaginal extirpation.

Fibromata of the Ovary.—ROVTIER (*La Gynécologie*, No. 6, 1901) reports six cases in his own practice, in all of which he diagnosed a solid tumor, but was unable to determine whether it was of ovarian or uterine origin. This doubt, he believes, should not influence the surgeon's decision, since the indication is clear to remove a pedunculated growth by the abdominal rather than by the vaginal route, in order to preserve the uterus.

In the cases reported ascites was noted only once, rather an exception to the fact that ovarian fibromata are usually accompanied by hydroperitoneum. It was evidently due to irritation. The patient was sixty-two years of age, but the five others were under thirty-six.

The benign character of these growths is shown by the fact that they are unilateral. Menstruation may or may not be affected, both menorrhagia and amenorrhœa being observed.

As in the case of all pedunculated abdominal tumors pain is variable, and seems to be due purely to mechanical causes.

Plastic Operations for Prolapsus.—BAUMM (*Centralblatt für Gynäkologie*, No. 21, 1902) reports 86 cases from the Breslau clinic, extending over a period of three years. Recurrence occurred in 30 per cent. The writer performed amputation of the cervix with colporrhaphy and perineorrhaphy, but made no attempt to keep the uterus anteverted, believing that it makes no difference whether the organ is forward or backward, provided that the prolapsus is relieved. The after-results of vaginal fixation do not commend the operation to him.

Atmokausis in Hæmophilia.—PINCUS (*Centralblatt für Gynäkologie*, No. 22, 1902) discusses the question whether "bleeders" ought to marry, which he answers positively in the negative. In order to obviate the necessity of enforcing this rule, he suggests that at the first appearance of the menses in a young girl, with such a loss of blood as to threaten life, the uterine cavity should be obliterated by atmokausis. If the patient survives the profuse monthly bleeding and marries a second indication may arise after labor or during the puerperium. At the climacteric in such a patient the indication is clear.

Intra-abdominal Pressure.—MEYER (*Centralblatt für Gynäkologie*, No. 22, 1902) considers this expression an erroneous one, and affirms that there is no such thing as a constant pressure which is the same at every point in the peritoneal cavity. The comparison between the difference in pressure in the pleural cavity and in the external air and that within the peritoneum is not justified on account of the elasticity of the abdominal walls. In the normal condition the equilibrium between the abdominal cavity and its contents is maintained, and when the air is allowed to enter at the operating-table the intestines, even when not previously emptied, protrude only to a slight degree. If they have been thoroughly evacuated it is even possible to establish a negative intra-abdominal pressure artificially by drawing up the unopened peritoneum, the muscular wall being thoroughly relaxed under anæsthesia Normally it cannot exist. That the pressure is never constant at all points is evident when one considers the variable conditions present in

the intestines and bladder, the peristaltic movements and those of the diaphragm, the contractions of the voluntary muscles, etc. The supposed variation in pressure due to different positions is also entirely theoretical.

Blood Examinations in Pelvic Suppuration.—LAVBENBURG (*Centralblatt für Gynäkologie*, No. 22, 1902) repeats the observations of Ditzmann, with whose conclusions he agrees, adding some interesting deductions of his own. He finds that in cases of suppuration there is a diminution of the red corpuscles, with a rapid increase in the white. The latter gradually diminish in number after the acute stage, to increase with each exacerbation. The leucocytosis varies directly according to the intensity and extent of the suppurative process. If it is limited the number of white corpuscles soon sinks to normal; in protracted febrile cases the leucocytosis varies with the different phases of the disease, the red corpuscles behaving in the opposite way.

The influence of the patient's constitution upon the leucocytosis is quite striking. In a subject who is exhausted by a long illness, or by loss of blood, an increased leucocytosis may persist even after the pus focus has disappeared.

The possibility of mistaking true leukæmia for temporary increase in the white corpuscles must be borne in mind. The increase in eosinophile cells and the presence of large mononuclear cells with neutrophile granules, not seen in normal blood, would settle the diagnosis. Moreover, it must not be forgotten that increased leucocytosis may be present in connection with wasting diseases, fevers, cancer, etc., and that there are daily variations, especially during digestion.

Cystic Degeneration of the Ovary.—KAHLDEN (*Beiträge zur path. Anatomie u. zur allgem. Pathologie*, Band xxxi.) presents the results of his careful studies of an old subject, on which he throws new light. He divides the cases into three groups, viz.: 1. Those in which the ovary is filled with small cysts, showing various stages of degeneration, where it is difficult to decide macroscopically whether the condition is normal or pathological. It seems to represent essentially an increase in physiological processes. 2. In this group the changes are more evident, as some of the dropsical follicles attain a considerable size and others atrophy. Hyperæmia is more marked, with resulting hemorrhages, so that some follicles lose their lining epithelium and are filled with coagula. 3. In the third group the cystic formation is general, though ova can still be found in some of the follicles, even when they are denuded of epithelium. Degeneration of the ovum is often preceded by changes in the membrana granulosa.

Microscopically, the writer found that in the majority of the cases so-called *hydrops folliculi* was due to glandular formations following ingrowth of the germinal epithelium, and not to simple dilatation of a pre-existing follicle. Moreover, he demonstrated the fact that this mode of origin could be inferred in some cases of ordinary cystic degeneration. [The practical bearing of these observations will be evident when one recalls the frequency with which ovaries used to be sacrificed merely because a few cysts were observed on their outer surface. While this condition is no longer regarded as a proper indication for removal of an ovary, we may well ask whether the pendulum

has not swung too far in the opposite direction, and whether "conservative surgery" has not been practised in cases in which any interference with the normal organ was mischievous. We have always looked with disapproval upon ignipuncture and the excision of small dropsical follicles, and the custom of including such cases under the head of true conservative operations.—H. C. C.]

Radical Operation for Cancer of the Uterus.—In a recent discussion before the Dutch Gynecological Society (*Centralblatt für Gynäkologie*, No. 22, 1902) the consensus of opinion seemed to be against the abdominal operation. Kouwer affirmed that it had not been proved that it was radical in the sense of removing all the disease. He did not believe that it was often possible to tell whether the lymph nodes were affected or not. Of thirteen patients operated upon by himself within the past three years by the abdominal route, five died from the operation, two had ureteral fistulæ, and two had an early recurrence. Treub thought that with a mortality of 33 per cent. simple palliative treatment was preferable.

Treatment of Inoperable Cancer with Methyl-blue.—CUCCA and UNGARO (*Rassegna d'ost e gin.; Centralblatt für Gynäkologie*, No. 22, 1902) use the following solution: Methyl-blue, 90 grains; 90 per cent. alcohol and glycerin, āā 3 drachms; water, 7 ounces. This is applied to the diseased cervix on tampons after previous curetting. A weaker solution is used for vaginal and intrauterine irrigation. The results have been quite satisfactory, patients being kept comfortable and free from hemorrhage and discharge for months, or even years. Pain was relieved so that morphine could be dispensed with, and the progress of the disease was evidently retarded. No unpleasant effects were noted after long use of the remedy.

Hæmatocele following Divulsion.—CARSON (*Progrès Méd. belge*, No. 1, 1901) reports the case of a young woman in which divulsion and curettement were performed for menorrhagia. Three days later she had abdominal pain, elevation of temperature, vomiting, and tympanites. A hard tumor appeared on the left side, extending from the cul-de-sac to the umbilicus. As septic symptoms developed, a vaginal incision was made and a quantity of pus and blood was evacuated, the patient making a good recovery. The reporter suggests that there might have been a rupture of the veins of the pampiniform plexus. [Why not rupture of the cervix or perforation of the uterus?—H. C. C.]

Menorrhagia in Young Girls.—SIRDEY (*Révue Gen. de Chir. et de Therapeutique; Der Frauenarzt*, 1901, No. 8) refers to cases of obstinate menorrhagia in which no local cause can be found. In consequence of modesty and ignorance the patient often becomes exceedingly weak and exsanguinated before the physician's attention is called to the condition, or else she is wrongly treated for anæmia. The flow is only increased by the tonics which are given, if no change is made in the hygiene or environment.

As regards treatment hot douches are seldom necessary. Hot rectal irrigation may be substituted. The patient should be kept absolutely quiet in

bed until a day or two after the flow has entirely ceased. In the intervals between the periods all violent exercise—dancing, cycling, etc.—should be interdicted, as well as balls and theatre-going. Cold douches and massage are beneficial. The diet should be simple, stimulants, tea and coffee being forbidden. Ergot and hamamelis are recommended during the flow.

OBSTETRICS.

UNDER THE CHARGE OF

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Suppression of Urine following Labor.—McKERRON (*Journal of Obstetrics of the British Empire*, 1902, vol. iv. No. 1) contributes a paper upon this subject, with a report of six cases from his own observations.

His first case was that of a multipara who gave premature birth to a still-born fœtus at the eighth month. The urine had been examined at intervals of about a month, but showed no albumin. There is no statement regarding the specific gravity, quantity, percentage of urea, nor whether casts or epithelial débris was present. During her first pregnancy she had had œdema, scanty urine, with a large quantity of albumin. She had been delivered of a dead fœtus at seven months, after an easy labor. During the pregnancy under consideration she remained fairly well until she was seized with sudden pain in the lower part of the abdomen, followed by vaginal hemorrhage. This ceased, but a few hours afterward a seven-and-a-half months' fœtus, but recently dead, was suddenly expelled. The placenta was covered with adherent black clots. Following this labor the patient had complete suppression of urine, which very gradually gave place under appropriate treatment to a temporary polyuria. The patient made a gradual and apparently complete recovery. Complete suppression of urine lasted in this case about three and a half days. McFerron states that it is the sole recorded case which he has found in which recovery followed complete suppression of urine after labor.

The second case was that of a multipara who had much sickness and prostration during pregnancy, who had borne one living child and had lost two children at the sixth month. During the pregnancy in question the urine was not examined, because the symptoms were similar to those in previous pregnancies when the urine was found to contain no albumin. This patient was suddenly seized with severe pain in the epigastrium and discharge of blood from the vagina. She expelled a dead fœtus, had suppression of urine, and died on the fourth day.

Case third was that of a primipara who was under observation until she entered the hospital. She was delivered by forceps of a living child. This

was followed by partial suppression of the urine, from which she slowly recovered. It is thought that her improvement and recovery were largely due to her delivery instrumentally under anæsthesia.

Case fourth was delivered prematurely in her third confinement of a stillborn fœtus. She had suppression of urine, and died on the eighth day. This case is quoted, and did not come under the observation of the writer.

Cases fifth and sixth are quoted from the practices of others, case fifth being that of a primipara with stillborn child, and said to have had no symptoms during pregnancy. She died on the fifth day after delivery. Case sixth had suppression of urine following premature delivery at the seventh month. The child was stillborn, and the mother died on the fourth day.

(We are especially interested in the cases seen by the writer. The type of case is not at all an uncommon one—namely, of an acute and overwhelming toxæmia. It is extraordinary to notice that in few if any of these cases the patient had received careful observation during her pregnancy. In one case the urine had not been examined, because the symptoms were like those of a previous pregnancy in which the urine was examined. The disposition is to lay almost entire stress in examining the urine of pregnancy upon the presence or absence of albumin, and this has been shown to be a most unworthy reliance.)

The writer concludes from these cases that a pathological condition of the kidneys was present, resulting from pregnancy, but in some cases dependent upon a pre-existent defect of the kidneys. The question of the condition of the liver does not enter into his calculation.

He recognizes the influences of a neurotic temperament as producing hysterical suppression of urine, and infers that this may bring about the dangerous and fatal issue seen in these cases. In none of the fatal cases is there a report of an autopsy.

In the matter of treatment he draws attention to the injection of saline solution into the rectum. He believes that opium in the form of morphine does not further the suppression of urine, although he states in his closing sentence that opium should be rigidly withheld in cases where anuria is to be feared.

[While these cases are interesting as clinical reports, they are incomplete from the faulty or deficient study of the urine during pregnancy, the lack of post-mortem examination, and the failure to completely study essential phenomena of toxæmia. While the injection of saline fluid into the rectum is valuable, it is inferior in prompt and direct effect to intravenous saline transfusion or even to hypodermoclysis. Threatened death from toxæmia is so grave a condition that it fully justifies transfusion or any other rational procedure.]

Spontaneous Expulsion of a Dermoid Ovarian Cyst during Labor.—HAULTAIN (*Journal of Obstetrics of the British Empire*, April, 1902) reports a case of this rare condition and quotes four cases from the literature of the subject.

The writer's case was that of a woman in her third pregnancy who had been in labor for twenty-four hours. A slight swelling bulging through the posterior vaginal wall delayed the exit of the child. An attempt to deliver by forceps

had caused a mass to protrude from the vulva, which was pushed up into the vagina. Labor pains then ceased. On examination the mass was found in the vagina, and on pulling it down for inspection it became detached and was removed. It resembled an œdematous polyp about the size of a small cocoanut. The child was delivered with forceps without difficulty. On examination a rent was found in the posterior vaginal wall, through which the tumor had made its way. The tumor was examined and found to be a dermoid cyst containing hair and sebaceous material. The rent in the vagina was packed with iodoform gauze. The patient made an uninterrupted recovery, the gauze being gradually removed.

Cardiac Disease in Pregnancy.—Under this title JARDINE (*Journal of Obstetrics of the British Empire,* April, 1902) publishes notes of thirteen cases in which cardiac disease has complicated labor. Among these there are three cases of aortic stenosis, one of which terminated in premature labor and two in labor at full term. The mother made a full recovery, and two of the children lived. The remaining cases were those of mitral disease, in which the degree of incompetency varied, as did also the condition of the heart muscle. In some of the cases mitral stenosis was present, and two cases were complicated by tricuspid incompetence as well. Most of these patients went to full term. There was one death from cardiac failure. One patient had high fever, which fell after the bowels had been thoroughly moved, the patient discharging a large quantity of very offensive feces. Another patient after labor was seized by a rapidly increasing and most threatening anæmia, from which she made a very gradual recovery.

Jardine calls attention to the low death-rate in these cases—1 to 13. He calls attention to pure mitral stenosis as being the most deadly form of heart disease complicating labor. When labor ends in these cases the uterine vessels are cut out of the circulation, and if there has not been a free hemorrhage during the third stage of labor, blood is returned to the right side of the heart, which may become engorged and paralyzed. Hence, bleeding should be encouraged during the third stage of labor, and if it does not occur from the uterus, and the patient's condition is threatening, blood must be taken from a vein. Attention is called to Hart's illustration of a case of this sort, showing the distention of the right auricle.

Aortic incompetence is next in gravity, and mitral incompetence if the heart muscle is in good condition is less dangerous. Cardiac lesions do not show themselves during pregnancy until after the middle of this time. Then breathlessness, palpitation, cough, and œdema appear, with albumin in the urine. The patient may not be able to lie down.

The woman who has heart disease and who has suffered from failure of compensation at any time should be strongly advised not to marry. So far as treatment is concerned during pregnancy, the patient must be kept at rest, the bowels moved freely, the kidneys stimulated, the lungs relieved, and cardiac tonics freely given. Strophanthus is a better drug than digitalis in these cases. While the patient will probably improve, the cardiac tonic must be continued.

In a bad case the induction of abortion before the fourth month is permissible. In the later months labor should not be induced.

When labor begins large doses of strophanthus or digitalis should be given. As soon as the os is fully dilated labor should be terminated. The patient should never be allowed to bear down. If the heart begins to be embarrassed before the os is dilated the os must be stretched open or incised, chloroform given, and delivery at once effected. The uterus should be allowed to relax and bleeding encouraged after the birth of the child. Ergot is contraindicated unless in exceptional cases. If the patient did not bleed from the uterus and showed embarrassment blood should be taken from the arm. During the puerperal period free stimulation and cardiac tonics should be used. When patients die after labor it is from acute œdema of the lungs and cardiac failure. Nursing should be prohibited, as it influences the heart badly.

Blood-counts in the Newborn.—AITKEN (*Journal of Obstetrics of the British Empire*, April, 1902) contributes an interesting paper upon this subject based upon 300 counts and 100 stained films in 43 different infants. The weights of 200 children are also utilized for purposes of study. Aitken finds that the red corpuscles of the blood of the infant at birth are relatively more numerous than in the adult. During the first days of life, when the child is losing weight from urine, perspiration, and is fasting, the number of cells increases. From the second day the number gradually falls, so that by the tenth day the red cells are less numerous than at birth. During this time the child gains in weight. Soon after birth the corpuscles vary in size and shape, and there is deficiency in rouleaux formation. Nucleated red corpuscles are present at birth and as late as the ninth day after. Their numbers vary greatly. Hæmoglobin is relatively higher at birth than immediately afterward, usually over 100 per cent., individual cells being richer than those of the adult. At birth the white corpuscles are two or three times as numerous as in the adult, increasing during the first forty-eight hours of life. After the third day the number of white cells diminishes, and by the tenth day it is much less than at birth. After the first feeding leucocytosis accompanying digestion is very marked, and after a fast in the later days. At birth the lymphocytes exceed the neutrophiles. After the first feeding the latter increase considerably. This gain diminishes as the effects of digestion wear off and the blood becomes less concentrated, until in the second week, the lymphocytes are again increased as at birth. The percentage of eosinophiles is greater than with adults.

Ovarian Tumor Complicating Advanced Pregnancy Removed by Section.—In the *British Medical Journal*, April 5, 1902, DUNNE reports the case of a patient in her fourth pregnancy who had suffered from a rapidly-growing ovarian cyst. The abdomen was greatly distended, measuring 42½ inches at the umbilicus and 42 inches at a point higher up. Two distinct swellings were visible separated by a well-marked groove. On the right side the tumor was soft and fluctuating and on the left the uterus was smaller and hard. Fœtal heart sounds were heard at a point midway between the umbilicus and the anterior posterior spine on the left side. The os was patulous and the fœtus could be palpated within the uterus, the head presenting. As labor was imminent, operation was performed at once. The abdomen was opened on the right of the umbilicus. There were no adhe-

sions, and the cyst after puncture was easily drawn out, its pedicle was secured, and the abdominal cavity washed out with hot boric-acid solution. One-fourth grain of morphine was given hypodermically to delay labor, as uterine contractions were noticed during the operation. On the next day, when dilatation was complete, the membranes ruptured, and the child was delivered with axis traction forceps. The patient was not permitted to bear down. The placenta was removed digitally, and a copious douche of iodine solution given. Pressure was maintained above the pubes and a large pad and binder were applied. The intra-abdominal pressure was maintained by a tight binder over the dressings. The mother made a good recovery, but did not nurse the child.

OTOLOGY.

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The Significance of Lumbar Puncture in the Diagnosis of Intracranial Complications of Otitis.—IWAN BRAUNSTEIN (*Archiv. f. Ohrenheil.*, Band liv., Heft 1–2.) (Published from cases in Schwartze's clinic in Halle A. S.) For the lumbar puncture in the Halle clinic an ordinary hollow needle is used, without aspiration. The puncture is made just below the fourth lumbar vertebra, with the patient lying on the side, scrupulous asepsis being observed. The fluid is allowed to flow until generally about 30 to 40 c cm. are collected, as this quantity ensures that some of the fluid comes from the cranial cavity. Only the following points in regard to the cerebro-spinal fluid obtained are considered of diagnostic worth: 1. Clearness (normal cerebro-spinal fluid should be as clear as water). 2. Presence of leucocytes in the fluid (normal fluid contains very few). 3. Presence of bacteria in the fluid (cover-glass preparations, cultures, and animal inoculation are necessary to prove the sterility; normal cerebro-spinal fluid is sterile).

Unpleasant experiences are found to follow the lumbar puncture very rarely if the puncture is carefully carried out. Two cases of death following closely on the puncture are reported; one due to aspirating the fluid with a syringe after it had ceased to flow, and the other quite as likely due to the chloroform narcosis (autopsy showed fatty degeneration of the heart). Sixty-seven lumbar punctures carried out on 48 patients are reported. Of these, 6 were meningitis purulenta, 10 were meningitis purulenta with brain abscesses, 3 were meningitis purulenta with sinus thrombosis, 1 was probable epidemic cerebro-spinal meningitis, 1 was meningitis serosa, 7 were sinus thrombosis, 3 were perisinous abscess, 2 were sinus thrombosis with encap-

sulated meningitis and brain abscess, 4 were sinus thrombosis and brain abscess, 5 were tubercular meningitis, 6 were cases of intracranial pressure presenting meningeal symptoms.

Twenty-nine times the puncture gave normal fluid (negative result). Of these, 7 were in cases of sinus thrombosis, 3 were in perisinous abscess, 7 were in brain abscess, 5 were in sinus thrombosis with brain abscess, and 7 were in cases of intracranial pressure with meningeal symptoms. On the other hand, in those cases where a general purulent meningitis existed, either alone or as a complication, the cerebro-spinal fluid showed the above-described abnormal conditions. The cases of cerebro-spinal meningitis and meningitis serosa reported are so few in number that the conclusions drawn from them have little worth.

As to the tubercular meningitis, Braunstein believes that the opalescent cloudiness of the fluid gives a fair presumption that the intracranial trouble is meningitis tuberculosa, even when the microscopical examination fails to show the presence of the tubercle bacillus.

As to the diagnostic worth of lumbar puncture in intracranial complications of otitic diseases, Braunstein concludes:

1. Obtaining by lumbar puncture normal cerebro-spinal fluid, when from its amount you can conclude that a part comes from within the cranium, rules out with great certainty a general purulent meningitis.

2. Obtaining by lumbar puncture cerebro-spinal fluid which presents the above-described abnormal characteristics proves the presence of a diffuse purulent meningitis.

On the Pathology of Deaf-mutism and the Window Niches.—PROF. J. HABERMANN, of Gratz, divides deaf-mutes into two classes, hereditary and acquired. The percentage of each varies with different writers. Unchermann found in 1885 about 1841 deaf-mutes in Norway, of whom 51 per cent. were heredity, and the remaining per cent. were acquired, with the exception of 0.5 per cent., in whom it could not be determined. It is not always possible to determine, even by examination after death. Most cases of acquired deaf-mutism are caused by diseases of the labyrinth, most of which have spread from the brain or middle ear. Mygind, in his work in 1894, reported over 139 cases in which the middle ear only was diseased, but he stated nothing about the labyrinth or the histological examination of the labyrinth. Thus Matte could completely deny the occurrence of deaf-mutism due solely to middle-ear disease.

Habermann reports two cases in which the middle ear only was demonstrable as a cause of the deaf-mutism. He gives the gross and microscopical pathology. In the first case, a boy thirteen years, who died of general tuberculosis, two different pathological changes were found. First, a recent tuberculosis of the middle ear, which had nothing to do with the deaf-mutism. Second, in both middle ears changes of a chronic nature, which could have existed for some years. In the right middle ear great thickening of the lining, numerous adhesions binding down the ossicles, a bony closure of the round-window niche, and hyperostosis of the bone on the inner tympanic wall. On the right drum a scar, and on the left an old perforation and calcification were present—effects of a suppurative otitis media. This middle-ear sup-

puration must have occurred early in the first weeks or months of the child's life. No pathological changes were found in the brain or nervous mechanism of the internal ear. The auditory canal was normal. For the explanation of the deaf-mutism only the changes in the middle ear remain. Panse, after a study of all the cases in literature in which pathological changes were found in the windows, came to the conclusion that diseases of both windows caused a considerable degree of deafness. Habermann reported a case, with specimen, to the Otological Society in Dresden, where both windows, right and left ears, were closed by bone and connective tissue, with complete deafness on one side and nearly complete deafness on the other. The bony closure of the round window and the fixation of the stapes by bands of connective tissue is sufficient therefore to explain the deafness in this case.

The second case, a woman, thirty-eight years old, died of interstitial nephritis. The pathological changes have a similarity to those of the first case, and are also to be interpreted as a result of a suppurative otitis media—a great thickening of the lining membrane of the middle ear; hyperostosis and sclerosis of the bone, and almost bony closure of the round window by bone tissue. The left drum showed a large old perforation; the right drum was thin. The pustule on the left drum is hard to make consistent with the rest of the aural conditions.

Bleb formation and small abscesses in the drum are not unusual in certain forms of inflammation of the external auditory canal as well as in acute suppuration of the middle ear, with copious exudation in the tympanum; but in a chronic suppurative case with minimum exudation and a large perforation of the drum the bleb cannot be explained. We can only regard the general condition of the patient to account for these changes in the ear. The patient had uræmia, with neuroretinitis, albuminuria, and enteritis. It is known that patients with kidney disease are much inclined to inflammatory tissue changes. Thus diseases of the skin in Bright's disease are not rare, and in the beginning, according to Thursfield, patients have pruritus, urticaria, and eczema. In the final stages, and in anæmic patients, erythema, bullous, and desquamative processes; further, purpura and pus infections.

In consequence of chronic suppuration pus cocci were already present, and the uræmia of the patient explains the appearance of the pustule in the epidermis of the drum.

The results of the hearing tests of this case are important. Unfortunately the tests were taken late, and the patient was very weak. A part of the investigation is unreliable. The patient had nearly complete deafness on the left; heard the watch by contact on the ear; heard numbers loudly spoken 0.03 m.; also on stopping both ears. Right: numbers; voice, 1.20 m., and numbers, whispered voice, 0.15 m. As to these results no doubt can exist, and the assertion is, therefore, correct that the patient still possessed this acuteness of hearing with bony closure of the round windows and free oval windows.

Other writers have come to the conclusion that bony closure of the round window alone does not cause deafness. Mygind reports a case of a deaf-mute child who had some hearing in spite of bony closure of the round window, with masses of cholesteatomata pressing through the oval window into the

labyrinth. In the case described by me, could the objection be made that the window niches had not come to a complete bony closure, therefore, vibration of the membrane, at least of the posterior part, was yet possible? If we consider that the remaining space was not filled with air, but with a loose, wide-meshed connective tissue, and that these spaces were saturated with tissue fluids and completely closed off from the tympanic cavity by a new-formed thick bony wall, it is evident that a movement of the membrane outwardly by a movement of the labyrinthine fluid, due to the influence of sound, was impossible. This condition of the left ear makes it possible to consider the hearing acuteness of the right ear certain; then it is not a question of conduction from the nearly deaf left ear.

For good hearing the unhindered mobility of the membrane of the round window is necessary; that with bony closure of the round window one can even hear whispered speech close by; that with high degree of obstruction of the function of both windows speech-deafness had resulted in both ears in Case I. and in the left ear of Case II. From these cases and the case demonstrated in Dresden it is very apparent that under certain conditions deafness for the tuning fork by bone conduction can happen through complete closure of both windows.—"Zur Pathologie der Taubstummheit und der Fensternischen," *Archiv. für Ohrenh.*, 1901, Bd. liii., S. 52–67. Tafel I., II.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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Retention of the Pupil Reflex to Light after Intracranial Division of the Optic Nerve.—MARENGHI (Pavia) before the International Congress of Pathologists at Turin (*British Medical Journal*, October 26, 1901), stated as the results of his experiments, and demonstrated to the congress in a rabbit, that the iris of an animal whose optic nerve was divided at its point of departure from the chiasm responded to light; the reaction was slow and weak, but distinct. The result could be explained by the existence of a peripheral reflex centre within the retina. Further investigation is needed.

Primary Chronic Glaucoma.—LITTLE, London (*British Medical Journal*, October 26, 1901), in his Presidential address before the Ophthalmological Society of the United Kingdom upon some clinical experiences of primary chronic glaucoma and the value of iridectomy, lays down the rule that an operation should be done in every case in which the patient is strong enough

to stand it, and the earlier it is done the better; but he would never hesitate to operate on an eye even if the fields were contracted to fixation, provided that there was any vision worth saving; even in the premonitory stage he advocated operation.

Gonorrhœa and Diseases of the Eye.—LAWFORD, London, in a discussion on the relation of the gonorrhœa to diseases of the eye (*British Medical Journal*, November 2, 1901), after excluding purulent ophthalmia, considers the following ocular affection as known to occur in association with gonorrhœa; a form of mild conjunctival inflammation (not due to direct inoculation), scleritis and episcleritis, iritis and iridocyclitis, neuroretinitis. Suppurative keratitis has been described in severe pyæmic case; of these iritis is the best known.

The Diagnosis of Malignant Intra-ocular Tumors in Infancy.—SOURDILLE, Nantes (*Gaz. Med. de Nantes*, November 16, 1901), calls attention to the great difficulties frequently existing in making the diagnosis of retinal glioma. Transillumination should be practised in every case before surgical intervention. If a tumor is shown, and any doubt as to its nature be present, it becomes necessary to make a puncture and to examine histologically and experimentally (by inoculation) the elements of the neoplasm.

Malignant tumors of the retina are all gliomata, properly so called. Mistakes of diagnosis are frequent. These mistakes are masked under the specious name of "pseudoglioma," when enucleation shows that an entirely different disease is present; and "cryptoglioma" when the disease is overlooked. For a long time the amaurotic cat's eye was believed to be adequate for the diagnosis of glioma. This has been the source of numerous errors. The writer concludes from his own histological examinations, that of ten eyes enucleated for glioma three are found to be cases of pseudoglioma.

The amaurotic cat's eye is by no means pathognomonic of glioma; this is found in suppurating retinal hyalitis, purulent choroiditis, affections following infectious maladies of infancy, particularly the eruptive fevers, and, above all, tuberculosis of the intra-ocular membranes, the choroid and retina.

It has therefore been proposed to narrow the diagnosis of these tumors and to lay down an absolute rule following Lagrange; in glioma the eye is glaucomatous or tends to glaucoma; in pseudoglioma the tension is below normal and tends to phthisis. This rule holds in the majority of cases, but there are many exceptions. There are cases in which glioma evolves under the form of iridocyclitis instead of glaucoma, where there is no increased tension whatever. Other cases directly opposite occur presenting the syndromes of glioma, amaurotic cat's eye and increased tension without the existence of any tumor. Transillumination and puncture are alone adequate to assure the diagnosis.

Benign Gangrene of the Eyelids.—ROGER and WEIL (*La Presse Médicale*, No. 76, 1901) report the case of a healthy male adult who was attacked with idiopathic gangrene of the left eyelids. There was no preceding inflammation of the conjunctiva nor any lesion of the lids. The disease began insidiously in a spot of inflammatory œdema. At the expiration of four or five

days general symptoms made their appearance, fever at first high (39.5° C.), but lasting only four days. It was accompanied by feeling of weight in the head and tension in the face, but without actual pain. There was a saburral condition of the digestive tract. The urine was albuminous. There was no delirium nor marked depression. Gangrenous plaques made their appearance upon the reddish erysipelatous lids forty-eight hours after the outbreak of the disease. The mortifying plaques were blackish in color and of very irregular contours. Line of demarcation quickly formed, and repair proceeded very rapidly. Eschars separated in fragments. The upper lid was free thereof on the nineteenth day; the lower on the twenty-first day. Healing was complete less than a month after the outbreak. There was no atresia of the palpebral orifice, and the only permanent trace was a slight ectropion at the external angle of the eye.

This is a very rare affection, indeed. The authors were able to find but a single analogous case in literature. It seems to resemble the spontaneous gangrene of the external male genital organs described by Fournier, of which more than twenty cases have been published since attention was first called to it.

Bacteriological study of the fluid obtained by puncturing the œdematous region showed the presence of an anaërobic microbe having a decided pathogenic action upon certain animals (rabbits, guinea-pigs), inoffensive for others (rats and mice). In the former, sometimes lethal septicæmia was produced, in other instances local lesions, œdemas, or indurations, or cold abscesses. True gangrene never occurred; but this is generally the case with all gangrene-producing microbes which do not retain or rapidly lose the virulence acquired for man.

DERMATOLOGY.

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Soft Pigmented Nævi.—FICK (*Archiv für Dermatologie und Syphilis*, Bd. lix., Heft 111), who has studied the microscopical structure of eight pigmented nævi taken from various parts of the body, sums up the results of his investigation as follows: The pathological increase of pigment in a nævus is not the cause of the tumor formation. Nævus-cell formation and proliferation on the one side and increase of pigment on the other are two pathological processes running parallel courses, but also occurring separately. The nævus cells only contain pigment when they are found in close proximity to

melanoblasts, with which they are in many places in demonstrable contact. In many nævi, giant cells are present without the growth showing other evidences of excessive proliferation or inflammation. These giant cells are distinguished from the other nævus cells not only by their size, but chiefly through the deep staining of their nuclei and the presence of a basophile granular substance in their protoplasm. The latter peculiarity is not found in all, but in many of the giant cells. Processes from the melanoblasts penetrate between the epidermis cells, partly coalescing with them. These cells lie partly between the cells of the epidermis, partly in the boundary between the epidermis and the corium, partly between the nævus cells, and partly arranged around the vessel in the deep layers of the cutis. With regard to pigmentation, the nævus cells behave just as the epithelial cells. All the pigment demonstrable in the nævus is found in cells (epithelial, nævus, giant, and melanoblasts). The histological picture presented by pigmented, soft nævi speaks categorically for the correctness of Ehrmann's pigment theory.

Botryomycosis in Man.—Bodin (*Annales de Dermatologie et de Syphiligraphie*, 1902, No. 4) reports two cases of this curious affection which he has studied histologically and bacteriologically. The first case was observed in a woman, twenty-seven years old. The disease began as a small nodule upon the posterior surface of the right thumb at its base. For a month the little tumor remained stationary, then it began to grow rapidly and ulcerate. At its acme the growth was the size of a hazelnut, rounded and covered with small fissures. At its base there was a deep circular furrow, at the bottom of which was a smooth, short, rounded pedicle by which the tumor was attached to the skin. The tumor was excised with the scissors and a complete cure followed in a few days. The second case also occurred in a woman, and was precisely like the first, except that the tumor was situated upon the palmar surface of the first phalanx of the middle finger of the right hand. Histological examination of the tumors showed that they were composed of young connective tissue extremely rich in vessels running in all directions, having very slender walls, often consisting only of the endothelial layer. In the external parts of the tumor there were marked evidences of inflammation. Search for the mulberry-like masses supposed to be due to the parasite named botryomyces—the so-called castration fungus of the horse—was completely negative; nothing but an ordinary coccus taking the Gram stain was found. As the result of his study of these cases the author concludes that the micro-organism described under the name botryococcus, and which is claimed by Poncet, Dor, and Spick to be the cause of a peculiar neoplasm in man, is not a specific organism, but is identical with the yellow staphylococcus; and the botryomycoccic neoplasmata are only fleshy granulation tissue produced under the influence of the staphylococcus aureus.

Atoxyl, a New Arsenic Preparation, and its Employment in Diseases of the Skin.—Schild (*Dermatologische Zeitschrift*, April, 1902) reports his experience with atoxyl (meta-arsenacidanilid) in the treatment of various diseases of the skin in which arsenic has been found more or less useful.

This new arsenical preparation is a white, odorless powder with a weak saline taste, soluble in warm water up to 20 per cent., and contains 37.69 per cent. of arsenic. A series of chronic diseases of the skin, such as alopecia areata, dermatitis herpetiformis, sarcoma of the skin, dermatitis exfoliativa chronica, xanthoma diabeticorum, psoriasis, lichen ruber, were given hypodermic injections of a 20 per cent. solution of the drug, the dose varying from 0.04 to 0.2. In alopecia areata and dermatitis exfoliativa no result was obtained; but in a case of dermatitis herpetiformis a cure was followed in a remarkably short time. In a case of xanthoma diabeticorum improvement was noted after the fourth injection and complete disappearance of the eruption after the twenty-second. In three cases of psoriasis improvement was clearly apparent after five or six injections, but a complete cure was not obtained in any of the cases. Of fourteen cases of lichen ruber treated in this manner, nine were, at the time of writing, completely cured, and the remaining five were rapidly improving. In about 1500 injections no irritation or infiltration worth mentioning was observed. The author is of the opinion that atoxyl represents a decided advance in arsenic therapy.

PATHOLOGY AND BACTERIOLOGY.

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On the Multiplicity of Amboceptors and Complements in Hæmolysis.—H. WENDELSTADT (*Centralblatt f. Bakteriolog. u. Parasitenkunde*, 1902, No. 10, Band xxxi., p. 469), working on the lines suggested by Ehrlich and Morgenroth, again demonstrates the multiplicity of the amboceptors in hæmolysis. A goat was immunized against the red blood corpuscles of oxen, pigs, and sheep. After the serum of the goat had developed strong hæmolytic properties for red blood cells of the other three animals their red cells were added in turn to the goat's serum, and the mixture kept on ice. By this means the intermediary body (amboceptor) for each kind of red cell was bound to that cell, and the remaining serum was still hæmolytic for the other two varieties of red blood corpuscles. For the separation of the specific complements two methods were used: First, by heating the goat's serum to 49.5° C. from twelve to fifteen minutes it was rendered inactive for both oxen's and sheep's blood, but readily hæmolyzed pig's red corpuscles. The second method consisted in adding dilute hydrochloric

acid to varying amounts of goat's serum. Wendelstadt now found that the hæmolytic property of the serum for the different kinds of red blood corpuscles was not all lost at once, but disappeared for the different animal's corpuscles at different dilutions; that is, the complement for oxen's red cells was lost first, next that for pig's blood, and most resistant to the action of acids was the sheep's blood. That the complement and not the intermediary body was injured in these experiments was shown by adding small amounts of fresh complements to the mixture in which no hæmolysis occurred, when dissolution was rapidly complete. By this method Wendelstadt believes he can demonstrate the multiplicity and specificity of the complement.—W. T. L.

A Contribution to the Pathology of Glanders of the Lung.—W. G. MacCallum (*Beiträge zur Pathol. Anat. u. allge. Pathologie*, 1902, Band xxxi., p. 440) describes the lesions found in the lung of a man who died of glanders following an accidental infection with an attenuated culture of the bacillus. At autopsy the lungs were found to contain numerous small, grayish-red firm nodules, both over the pleural surfaces and throughout the lung tissue. Similar nodes were found in the spleen and kidneys. The bacillus mallei was recovered from the nodules in the lung, spleen, and kidney as well as from other situations. From a histological examination the author finds three stages in the development of the nodules in the lung.

In the earliest lesions the centre of the nodule is composed of masses of leucocytes, fibrin, epithelial cells, and débris, the alveoli immediately surrounding this central portion being filled with plugs of fibrin, and outside of this, again, the alveoli containing epithelial cells. Large numbers of bacilli were found in the central portion.

In the second stage the central necrosis was more marked, and the exudate in the surrounding alveoli showed beginning organization.

The last stage is represented by a complete central necrosis, practically surrounded by a capsule of organized connective tissue.

The author, from these studies, agrees with Wright, Ehrlich, and Johne, and considers the process as a localized necrotizing bronchopneumonia.

Observations were made further upon the process of organization in the exudate surrounding the necrotic nodule. Lindeman believed the connective tissue arose from the alveolar epithelium, and Aldnigt, Fraenkel, and others derived the connective-tissue elements from the alveolar walls alone; while Ribbert thought organization took place from the walls of the finer bronchioles. Still other observers consider the greater interlobular septa and pleura as the points of origin. The author found in this case that organization commenced usually by a growth of connective tissue from the alveolar walls, but occasionally the connective-tissue elements also arose from the walls of the small bronchi. In serial sections the fibrin plugs were found to extend from one alveolus into another, or from the finer bronchioles into the alveoli, and the process of organization was followed along the fibrin masses directly from one alveolus to another. Thus an entire system of alveoli might undergo organization, the connective tissue growing from the alveolar wall into the exudate only at one point, and thence extending from one fibrin plug to another.—W. T. L.

A Case of Epidermoid from the Callosal Region.—Blasius (*Virchow's Archiv*, 1901, Band clxv., p. 504) states that intracranial pearl tumors (cholesteatomata) are certainly of epithelial type (to use the term in a morphological sense). The question arises whether they are epidermal or entodermal in derivation. At Beneke's suggestion, Blasius seeks to differentiate the two by macerating in silver nitrate. The two types of reaction are seen in cases of cholesteatomata. In the one type the sharp and continuous linear impregnation suggests the reaction of true mucosa. In the other (found in a case of cholesteatoma affecting the callosum) Blasius finds an impregnation quite after the fashion of, say, *vernix caseosa*—a row of dots or lines, discontinuous, ill-spaced, and of uneven diameter. Thus, despite the absence of hair and glands from the tumor, the diagnosis of epidermoid is made; indeed, in virtue of their absence, the origin may be referred to a remote point in the history of the medullary groove. When the pearl tumor is of mucosa type (of entodermal derivation) it lies usually at the base, perhaps derived from the anlage of the hypophysis. Blasius notes the frequency with which a meningeal nidus is reported for these tumors, but is inclined to regard the association as incidental to the development of an originally intracerebral (non-meningeal) focus. The pial or dural attachments would on this hypothesis be due to the pressure-atrophy of true brain elements which lay between tumor and meninges.

Remarks are appended upon granular ependymitis and upon multiple herniæ cerebri.

Weigert has held focal loss of ependyma as the first step in the onset of granular ependymitis. Blasius suggests, further, a mechanical origin for this loss (multiple ruptures consequent upon internal hydrocephalus), and regards the outgrowth of neuroglia following as a case of scar formation rather than as a reawakening of hitherto confined idioplastic tendency of glia cells, as suggested by Weigert.

Blasius adds four new cases of multiple herniæ cerebri to the four already in the literature. All four occurred together with intracranial tumors, and as a consequence of internal hydrocephalus. The herniæ sometimes occur into Pacchionian granulations, but they have usually a wider and different distribution.—E. E. S.

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